Region

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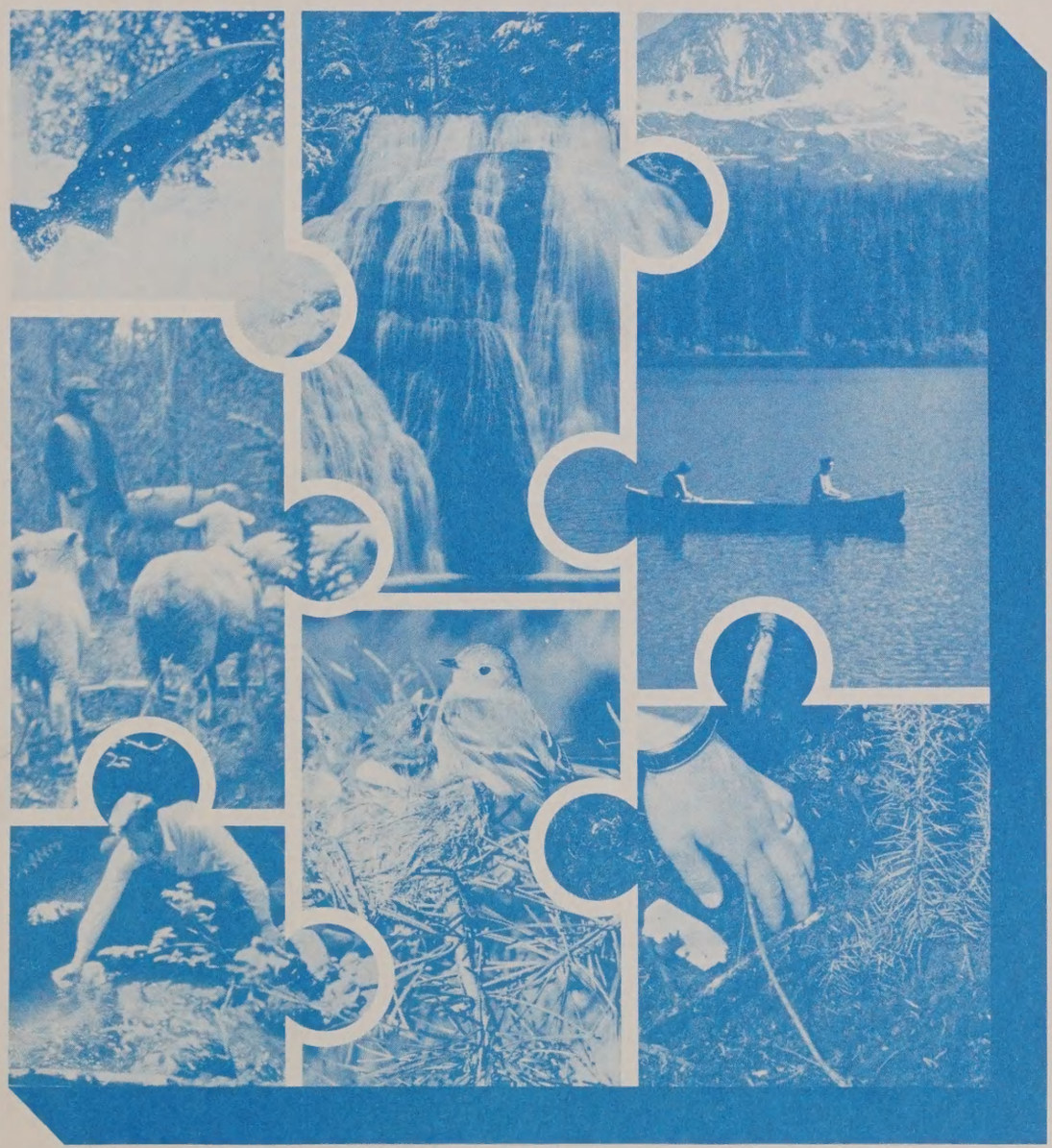
Managing Competing and Unwanted Vegetation

Final Environmental Impact Statement

Characterization and Management of Risk



Characterization and Management of Risk

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Introduction

Introduction

This document provides a comprehensive overview of the potential adverse human health effects associated with the vegetation management program proposed by the Pacific Northwest Region of the USDA Forest Service. It indicates the decisions made for managing the risks of those health effects and explains the reasoning in reaching those decisions. It is the culmination of 2 years of study by the USDA Forest Service, with assistance from the University of Washington, School of Public Health and Community Medicine, and Labat-Anderson, Incorporated, a private contractor in Arlington, Virginia, that specializes in conducting quantitative risk assessments. In addition many individuals, organizations, and federal and state agencies made valuable contributions throughout this effort.

The health effects were identified using the risk assessment process described in Appendix D and Appendix H, where detailed data are provided. The health risks are described in a number of ways and at various levels of detail. The degree of risk posed by the various vegetation management tools and methods is determined not only for forestry workers, but also for forest users and nearby residents. To the greatest extent possible, this document, supplemented by the other parts of the Final Environmental Impact Statement (FEIS), fully discloses any potential adverse human health effects of concern for routine and worst case situations.

Project History

The Pacific Northwest Region of the Forest Service was enjoined from the use of herbicides in its program of managing competing and unwanted vegetation; the Region was ordered by the court to conduct a worst case analysis in compliance with the National Environmental Policy Act (NEPA) of 1969.

Prior to the injunction, human health effects of herbicides were evaluated by individual foresters during project planning based on label instructions and information. In addition, water quality monitoring plans and emergency spill plans were developed for projects. Human health effects of other methods, such as the use of chainsaws, were considered at the time of project implementation through the use of job hazard analyses and tailgate safety sessions.

Since the injunction, the Forest Service, in cooperation with other State and Federal agencies, has evaluated, characterized, and made decisions about managing human health risks for all tools of vegetation management, including the use of herbicides. These risk evalu-

ations, characterizations, and management actions were conducted using the Environmental Impact Statement process prescribed by NEPA.

A Draft Environmental Impact Statement (DEIS) was issued for public comment in October 1987. The DEIS discussed how human health is related to the scope of the decision about the alternatives for vegetation management; to the alternative ways of managing competing and unwanted vegetation, and with regard to human health effects of methods.

In addition, human health was evaluated in two separate appendices. One appendix made rigorous quantitative estimates of effects using the best available information. The other appendix vigorously explored qualitative aspects of human health, again using the best available information.

Thousands of people commented on the DEIS. Most people commented on the human health aspects of the program. Scientific peer review of the human health portions was conducted by 11 scientists. All public comments and comments from the peer reviewers were considered, and changes were made to correct facts and improve the analysis contained in the FEIS.

Thus, the general public, interest groups, and cooperating agencies played a crucial role not only in defining the full dimensions of the human health issue, but also in providing information used in the evaluation and decision-making processes.

Approach to Evaluating Health Risks

In the course of evaluating risks and listening to public, agency, and scientific comments, it became apparent that there are two very different schools of thought in dealing with the risks associated with herbicides and other tools. One school of thought emphasizes what is known about human health effects and the track record of safe use of herbicides. The other school of thought raises troubling questions, emphasizes what is not known, and focuses on problems that have been experienced with the use of herbicides. There is merit in both schools of thought, and this document strives to present a balanced view that reflects both what is and what is not known.

There are three basic sources of information about the known and potential human health effects, that were used as the data bases for the risk assessment process:

(1) Studies of human populations (epidemiological studies), field studies of worker exposure, and historical information, such as State accident insurance records;

(2) Studies on laboratory animals; and

(3) Mathematical models based on detailed scenarios.

Each of the three approaches provides information about effects that might result from different kinds or levels of exposure to the hazards involved in vegetation management (sources 1 and 2 above) or about the levels of exposure that might result under specified conditions (source 3). These sources are particularly relevant to the evaluation of herbicide risks, and their use in this risk assessment is discussed in detail.

The Forest Service assembled all available relevant data, considered the implications of data they could not obtain, and made evaluations about risk management for their selected program alternative that were based primarily on what is known but that allowed for the implications of what is not known. The actions suggested for managing risk are cautious where the lack of data indicated some doubt about whether people might be affected. This strategy is most evident in the elimination from the program of two herbicides, diuron and fosamine, that did not have sufficient toxicological data to allow an evaluation of their risks.

The summary discussions presented in this document are organized into three major sections.

Document Overview

Section 2: Overview. In the overview section, the basic components of a risk assessment are presented, human health risks of the program are described in a general way, and conclusions about likely program health effects are given based on what is known about the tools and methods proposed for the Pacific Northwest Region.

Section 3: Risk Assessment. This section describes how handtools, mechanical equipment, prescribed burning, biological controls, and herbicides are used in vegetation management. It provides details about their possible short-term and long-term health effects, indicates what exposures and subsequent risks workers and members of the public might experience with each tool in the Region 6 program, and discusses the quality of the data on which these conclusions are based.

Section 4: Program Risks. This section summarizes the risks associated with all alternatives proposed for Region 6. It provides background data for viewing the human health risks of the Region 6 vegetation management program in the context of the human health risks experienced every day in the Pacific Northwest. It discusses in detail the program risk management, including mitigation measures, decisions on pesticide use, and monitoring requirements.

Overview of Risk Assessment

Because human health risks are a primary consideration in evaluating the vegetation management alternatives in the proposed Region 6 program, it is imperative to base any health risk management decisions on the best information possible. This section describes the process used by the Forest Service to generate the needed information and to use it to characterize program risks so that risk management decisions can be made.

The Strategy Used to Predict Human Health Effects

On a general level, the potential health risks associated with each of the vegetation management tools were derived in the same way. First, the way in which a given tool is used in vegetation management was established. Then available data were assembled that indicated what health hazards were associated with the use of that tool. This was followed by an analysis of the projected exposures to the tool by forest workers, forest users, and nearby residents. A quantitative risk assessment was then undertaken, based on combining the data on the potential health effects with the evaluation of likely program exposures.

For some tools (manual, mechanical), this was a fairly straightforward process. Injury records furnished the likely effects data, and proposed program acreage indicated likely exposures. For the herbicide method, the process was more complex because of the initial level of concern about health effects and the degree of uncertainty in information about each herbicide.

The best sources of data relating human health effects to pesticide exposure are epidemiology studies. These provide the most direct cause-effect evidence, but because studies exist for only 3 of the 16 herbicides, they could not be used to directly quantify program risks. Nevertheless, the studies that do exist, combined with evidence from poisoning incidents and the few laboratory studies on humans, suggest concerns about human health effects from the use of the herbicides. The quantification of risks depends on available studies in laboratory animals. Doses from animal studies were compared with doses that might be experienced by people exposed to an herbicide. These data are used in pesticide registration and are available for most types of toxicity endpoints for the 16 herbicides.

Quantification of program herbicide risks was based on two key numerical indicators related to the herbicides' toxic properties: (1) no observed-effect levels (NOEL's) seen in laboratory animal studies for

general toxicity and for reproductive toxicity; and (2) a cancer potency value if a herbicide showed increased tumor incidence with laboratory doses. The NOEL's were used as reference values for assessing the risks of small doses.

A variety of cancer causing models exist. The most conservative assumption, and the assumption we make here, is that even the smallest dose of a carcinogen is a hazard. The cancer potency value allows comparison between herbicides. The larger the number, the more potent the herbicide is believed to be. When used in a mathematical model that includes the expected lifetime doses to humans, the cancer potency number is used to estimate the risk to workers and the public of using that herbicide.

Another numerical indicator is the $LD_{50'}$ which shows the amount of material that could be fatal to the average laboratory rat. Experience has shown that if a dose to humans is within 10 percent of the $LD_{50'}$ it is very likely that poisoning will occur.

These indicator numbers were combined with estimates of possible program doses to workers and the public. The dose estimates were based on plausible exposure scenarios that were mathematical representations of the kinds of exposure that might occur with different herbicide applications combined with mediating variables, such as the herbicides' dermal penetration rate.

For general and reproductive effects, the dose estimate associated with a particular scenario was divided into the laboratory animal NOEL to derive an another numerical comparison called the Margin of Safety, (MOS). For example, if an herbicide NOEL was 5.0 based on animal studies and the scenario dose was 0.025, the margin of safety (MOS) would be 200. An MOS above 100 shows that an estimated dose is more than 100 times lower than a level showing no effects in a laboratory animal. A high MOS provides a high level of confidence that no human health effects would occur. In contrast, where an MOS is low (less than 100), the level of confidence that no human health effects would occur would also be low. MOS's were computed for workers and the public for all kinds of exposure, including accidents, for all 16 herbicides proposed for use.

The analysis of cancer risk was done only for those herbicides showing positive evidence (amitrole) or suggestive evidence (asulam, atrazine, bromacil, 2,4-D, 2,4-DP, glyphosate, and picloram) of causing tumor growth in the laboratory. The cancer risk was based on the chemical's potency multiplied by an estimated average lifetime dose. The risk had to vary between 0 (certainty that the herbicide would not

cause cancer) and 1 (certainty that an exposed human would develop cancer). The MOS's and cancer risks were the only numerical indicators of risk. The risks of other toxicity endpoints, including the risk of heritable mutations and possible synergism, were evaluated in a qualitative way.

The final phase of the risk assessment process was one of quality control. We carefully analyzed the quality of the data available for each herbicide. Were study designs appropriate? Were the right tests performed? Could study results be duplicated? Careful scrutiny was given the data bases underlying the conclusions regarding general systemic toxicity, mutagenic and carcinogenic toxicity, reproductive and developmental toxicity, immunotoxicity and neurotoxicity, and human epidemiology. The quality of the data underlying the quantitative risk assessment is indicated in the qualitative risk assessment shown in Appendix H, as well as summarized in the following descriptions. The data base was found to be sufficient to reach a decision for 14 of the 16 herbicides considered. Two herbicides, diuron and fosamine, did not have sufficient information, and should as a result not be used in the Region's program until sufficient information becomes available.

The risk assessment section that follows expands on each of these points for each proposed herbicide. The program risks section (Section 4) then uses the results of the risk assessment on all the vegetation management tools to compare the alternatives and define program risk management methods and strategies.

Components of the Risk Assessment Process

Understanding the human health risks of the Forest Service's vegetation management program requires definition of the important components of the risk assessment process: hazard evaluation, exposure evaluation, and risk evaluation.

- **Hazard** is the characteristic of an item or substance that renders it capable of producing injury or illness. Substances and items present more or less of a hazard. The degree of hazard is reflected in how careful one has to be to avoid injury or illness. Such substances as arsenic and such items as chainsaws are known to be hazardous. Such substances as water or sugar are known to be relatively less hazardous. Hazard evaluation identifies those characteristics for each program tool and describes them as adverse health effects in the following discussions.

- **Exposure** is the event during which the hazardous item or material comes in human contact. Exposures vary both in level or amount,

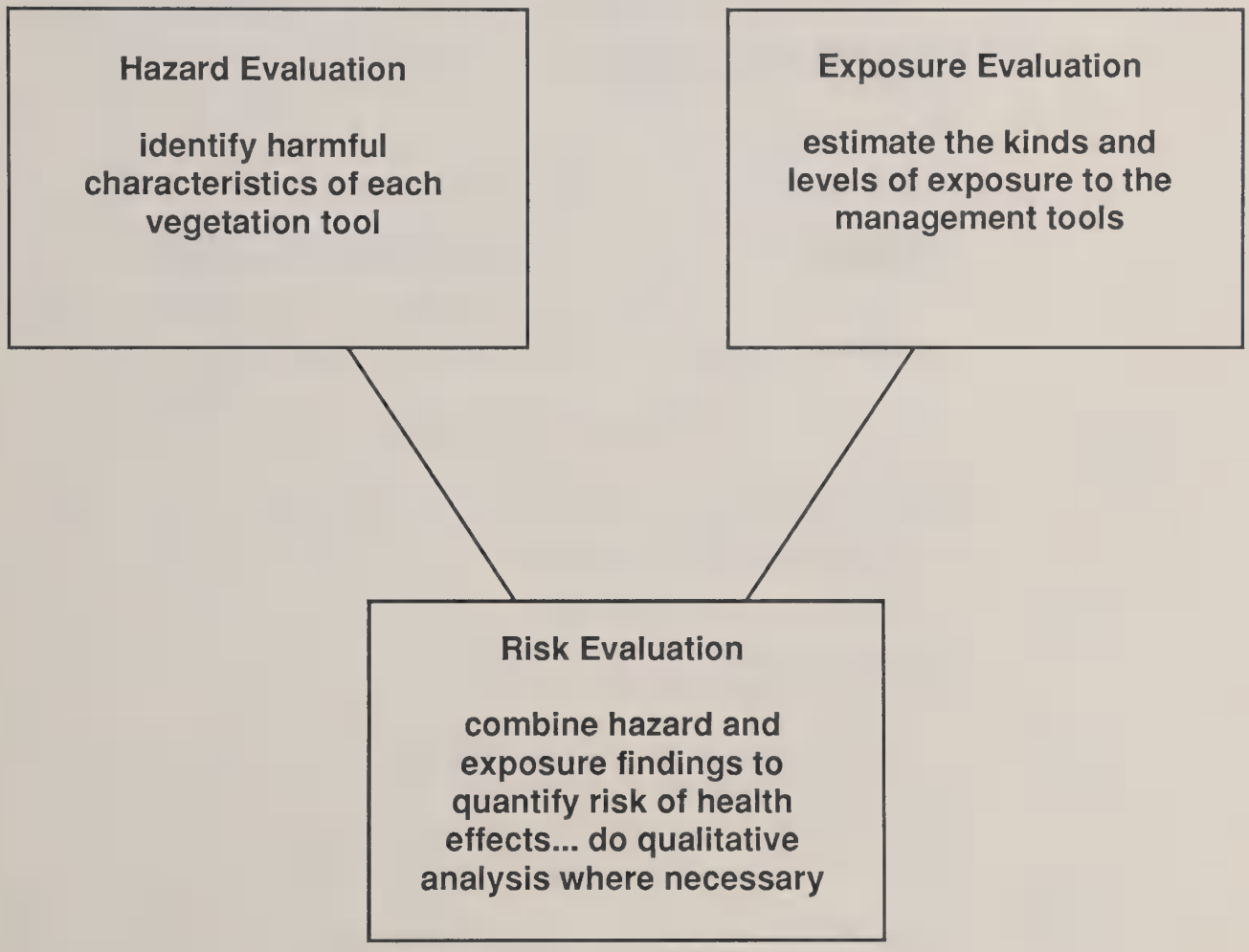
in duration, and in frequency. Exposure to such chemical substances as herbicides may lead to a dose, in which the substance is taken into the body through oral, dermal, or inhalation routes. It is the size of the dose and the frequency of exposure that determine whether a chemical causes health effects. A single, very small dose of arsenic may not produce any ill effects while a large dose of sugar may make an individual ill. Repeated small doses of some substances, even though they do not produce any immediate ill effects, may cause diseases, such as cancer, at a later period in time that become evident only after several years. Exposure evaluation estimates the frequency and duration of exposure to the program hazards. Doses for workers and members of the public are computed in milligrams (mg) of herbicide per kilogram (kg) of human body weight (usually assumed to be 70 kg for an adult).

- **Risk** describes the likelihood that a given exposure to an item or substance that presents a certain kind of hazard will produce an illness or injury. All human activities involve some level of risk because the human environment contains numerous hazards, including chemicals, tools and instruments, electrical appliances, motor vehicles, radiation, and physical obstructions. Risk evaluation uses the results of the hazard and exposure evaluations to draw conclusions about program risks.

Figure 2-1 illustrates the three risk assessment components. Government and private agencies have developed criteria for describing risks of exposures to chemicals in the environment in terms of those that would be acceptable. Principal criteria include cancer risks no greater than one in 1 million as indicating an undetectable risk increase over the background risk of cancer (FDA, 1984; EPA, 1986) and an MOS of 100 or greater (a 100-fold uncertainty factor) as indicating minimal risk of systemic or reproductive effects based on no-effect levels from chronic animal studies (National Academy of Science 1977, 1986). Those criteria are used in this risk assessment to evaluate herbicide risks. Potential program risks are broadly identified below. More detail on the hazards, exposures, and risks of the tools is given in Section 3 on risk assessment.

Figure 2-1

Components of the Risk Assessment Process



Potential Risks in Vegetation Management in Region 6

Potential program risks in vegetation management include the risk of injury from the use of handtools and mechanical equipment, the risk of illness or injury from exposure to smoke and fire in prescribed burning, the risk of waterborne disease from the use of grazing animals as biological controls, and the risk of illness from exposure to herbicides.

The immediate consequences of accidents from the use of handtools and mechanical equipment are well understood. However, use of these tools and equipment may produce such long-term health effects as ligament damage or exacerbate such conditions as arthritis.

Fire and smoke from prescribed burning and possible resulting wildfires present immediate risks of injury or death from burns and smoke inhalation and possible long-term effects, such as cancer or emphysema, from inhaling low levels of smoke for long periods.

Biological methods that use grazing animals in confined areas present the risk of transmission of disease organisms in fecal material through surface water runoff to points of human consumption.

Large doses of herbicides, such as those received by a worker in an accident, may present the risk of immediate (acute) effects. An example of such an accident is where a worker mixing herbicides spills concentrate on himself and does not immediately wash. Lower level doses repeated over time, such as those received by a worker whose main job is the application of herbicides, may present the risk of less severe long term (chronic) effects.

Exposures to the public from routine operations or accidental spills into drinking water - while significantly less than workers' exposures - none the less present some risk.

Effects to human health from exposure to herbicides depend on the dose received. Depending on the amount of the dose, these effects may be reversible or permanent and may include damage to specific organs or have localized effects on the skin or eyes.

Conclusions

Based on an analysis of the potential risks using the risk assessment process, both workers and the public potentially face several hazards associated with the Region 6 vegetation management program. To some degree, the risk from these hazards can be controlled by minimizing exposure via the management method used and the number of acres treated. In some cases, a quantitative risk assessment may be in quetion if there is a problem with the quality of data. The quality of the data may be in question if there is a problem with study design, duplicating the laboratory results, or the study has not yet been con-

ducted. In such cases, there is uncertainty regarding the potential health effects and the level of risk involved.

In all cases, workers are at greater risk than members of the public. Worker injuries are liable to occur in manual and mechanical vegetation management; members of the public should not be affected. Workers may suffer injuries, burns, and acute effects of smoke inhalation in prescribed burning and in escaped wildfires. The public is at lower risk because severe effects to them could result only in the case of a wildfire. They may also experience low-level effects from smoke, such as eye or bronchial irritation. There is some low-level risk of cancer from carcinogenic constituents in smoke. The cancer risk from smoke is not likely to be greater than one in 1 million for members of the public.

Some of the herbicides may present risks in terms of systemic and reproductive effects and in terms of the possibility of causing cancer, heritable mutations, and neurotoxic or immunotoxic effects. A number of steps have been taken to reduce these risks to acceptable levels, including eliminating three herbicides (fosamine, diuron, and amitrole) from the program and restricting application methods for many of the herbicides.

Our judgement is that cancer risk to the public from the program considered here is insignificant and indistinguishable from cancer risks to which the public is generally exposed. Public risks of cancer from the program are estimated to be less than one in one million.

Characterization and
Management of Risk

Risk Assessment

Risk Assessment

For each vegetation management tool, this section quantifies to the extent possible the health risks based on available data, discusses the quality of the data used to quantify risks and suggest human health concerns, and describes the potential human health effects that result from implementation of the vegetation management program. The discussion of herbicides, in particular, discloses where deficiencies and lack of toxicity information exists. Uncertainty as a result of incomplete information is also discussed as it relates to the characterization of risk.

Manual Methods

Use of Manual Methods

Manual methods use hand labor to remove competing vegetation or noxious weeds or to create conditions favorable to a desired plant's growth. Techniques include removing brush with chainsaws, pulling weeds by hand, girdling stems, and scalping soil. Attachment 1 of the summary is a profile of manual vegetation management tools.

Adverse Health Effects of Manual Methods

Working with such handtools as axes, brush hooks, machetes, and chainsaws can be hazardous under any circumstance. In forestry work, where site conditions can be extreme, handtools can be an even greater hazard. The risk of injuries increases if the size of the work crew increases or if the crew's work is required to be done in a relatively confined area. Worker fatigue can also increase the risk of injury. Workers could be cut by their tools, hit by falling brush, or fall onto the sharp ends of cut stumps or brush. Injuries can range from minor cuts, sprains, bruises, or abrasions to severe injuries that include major arterial bleeding or compound bone fractures. Unusually severe injuries may cause fatalities.

When temperatures are high, workers may experience increased fatigue, heat exhaustion, or heatstroke. Falls or other accidents may adversely affect pregnant female workers. Continued work in rugged terrain may initiate or exacerbate chronic health effects, such as ligament damage or arthritis. In extreme cases, exertion from manual methods in rugged terrain may bring on a heart attack or stroke in workers who are prone to such health effects. In addition, workers could be exposed to poison oak, ticks, and poisonous snakes.

Exposures to Handtools and Equipment

The likelihood of incurring injury appears to directly depend on the amount of worktime as described in the next section on the risk of manual method. Labor requirements vary with the type of work being done. Stavins et al. (1981) reported that labor requirements for Federal agencies in the Pacific Coast States were 2.5 to 5.0 person-days per acre for clearing and 0.75 to 3.125 person-days per acre for spot release treatment ranging from 25 to 100 percent of the brush being cut. The average was 3.75 days per acre for 100 percent clearing and 2.65 days per acre for all release treatments.

Quality of Information on Health Effects

The association between the health effects of manual methods and the use of the tools is fairly straightforward. For long-term health effects from the use of these tools, the associations may not be as evident, nor are records kept that would support these associations. Nevertheless, the relationship between hours worked and injury frequency appears to be a reliable one, so the quality of data is considered fair to good. The impact of the confounding variable, job experience, is not factored into the analysis.

Risks of Manual Methods

Up to the present, worker risk of minor and major injuries from manual methods in terms of the likelihood of injury per person-day worked has not been formally calculated. Several studies provide data for making estimates. Bernstein (1979) reported a frequency of one minor injury per 13 person-days with no major injuries, based on 265 person-days of brush removal. The same crew sustained a rate of one injury per 25 person-days during precommercial thinning. Roberts (1980) reported only minor accidents in 30 person-days of brush control with one near-miss of a serious eye injury. The Northwest Forest Workers Association experienced 29 hours of lost-time injuries per 100,000 hours worked and 47 reported injuries per 100,000 hours worked.

Brush-cutting injuries reported by the Bonneville Power Administration (1976-70) ranged from 2.5 to 10.3 injuries per 200,000 person-hours worked (U. S. Department of Energy 1983). Dost surveyed the Oregon State Accident Fund records for 1978-1979 to assess injury rates associated with forestry practices. Among the major items reported were strains and bruises (46.6 percent of reported accidents), eye injuries (8.6 percent), chainsaw cuts (7.0 percent), poison oak reactions (5.7 percent), fractures (4.8 percent), and vehicle and equipment accidents (4.8 percent) (Dost 1981).

Newton and Dost (1984), using data from the Oregon State Accident Insurance Fund (SAIF) and estimates of the number of acres of Oregon forests cleared annually by manual methods for 1978-1983, calculated that one accident occurs for every 130 acres cleared manually (per 438 person-days of work). The estimate from this study is used for the calculation of worker injuries for manual methods under each alternative.

Members of the public are not at risk from manual methods. The tools are used by the workers, and no member of the public is likely to come near enough to be injured when such tools are in use.

Conclusions

Minor injuries are almost certain to occur with the use of handtools. Severe injuries may occur but at a much lower frequency. One human health effect of concern is the serious injury that can potentially result from the use of chainsaws. These injuries can be mitigated with such precautions as appropriate training, scheduled rest breaks, and equipment maintenance and repair.

Mechanical Methods

Use of Mechanical Equipment

Mechanical methods involve the use of tractors, graders, or cable systems to remove unwanted vegetation. Equipment attachments may include blades, mowers, brush cutters, or plows for site preparation, roadside brush control, or removal of unmerchantable material. Attachment 2 of the summary is a profile of mechanical vegetation management equipment and methods.

Adverse Health Effects of Mechanical Methods

The operator and other workers in the vicinity of the mechanical equipment are at risk of injury. Serious injuries could result if the operator loses control of the machine on steep terrain. Such accidents are uncommon among experienced operators, but they are difficult to avoid entirely. Accidents may occur when pushing brush under conditions of poor visibility, when encountering a short headwall or road cut, or when misjudging the slope. If the machine overturns, it could seriously injure the operator, as well as create flying debris that could pose a hazard.

Workers could be struck by falling trees or by debris thrown by the equipment while it is operating. In such cases, the operator's assistant on the ground is at greater risk than the operator, especially when brush cutters or mowers are being used. A member of the public who

enters an area where mechanical equipment is being used could also be injured by flying debris. Injuries also can result from working around large machines that tend to be slippery, oily, or otherwise capable of contributing to operator risk during service or repair. In addition, the high noise levels associated with the operation of heavy equipment may cause hearing impairment, such as a temporary hearing threshold shift for the public or permanent hearing loss for a worker.

Exposures to Mechanical Equipment

The equipment operator and ground crews are the only individuals liable to be exposed to injury from mechanical equipment. Members of the public are not likely to be in close enough proximity to the machine to be injured. The worker exposures would vary depending on the program acreage to be managed mechanically.

Quality of Information about Mechanical Method Health Effects

The quality of data on mechanical methods health effects is lower than that existing for manual effects. There is an intuitive relationship between equipment use and injury rate but no real evidence from forestry use on which to base that conclusion.

Risks of Mechanical Methods

Little data exist to assess risks from mechanical methods of vegetation management. However, an assessment of their impact was made by Newton and Dost (1984). The most serious accidents associated with mechanical methods involve the overturning of machinery. Other risks are associated with rolling or snapping vegetation. According to the authors, only four accidents associated with mechanical clearing were reported to the original SAIF in 1979. The accidents were apparently not too severe, as they averaged less than $300 per claim. There was no acreage base available for these accidents. From these data it can be assumed that, while accidents are certainly a possibility with mechanical methods, both major and minor accidents are rare.

The risks to the general public from mechanical methods are very low. The only injuries to the public that are remotely possible from these methods are accidents resulting while the equipment is in transit from storage to the treatment site or in the extremely rare instance when a member of the public ignored safety precautions and entered a treatment site while the equipment was in use.

Conclusions

Minor injuries are almost certain to result from the use of mechani-

cal equipment. Severe injuries are likely to be rare. A health effect of concern is the severe injury or fatality that might result from the overturning of heavy equipment. Special precautions instituted to minimize the likelihood of this occurring include appropriate training, scheduled rest breaks, regular equipment maintenance and repair, and warning signs for the public.

Prescribed Burning

Use of Prescribed Burning in Vegetation Management

Prescribed burning methods include broadcast burning of forest residues using drip torch or helitorch on a clearcut to prepare a site for planting, pile burning of unmerchantable material from cuttings using drip torches or remotely ignited gelled gasoline packets, and underburning beneath tree stands to set back unwanted vegetation or promote desired forage or browse species. Attachment 3 to this risk summary profiles prescribed burning methods.

Adverse Health Effects of Prescribed Burning

Short-Term Effects

Prescribed burning presents the combined hazards of fire and smoke to ground crews at the site. Effects on workers may range from eye irritation, coughing, and shortness of breath in moderate-to-heavy smoke to severe burns that may result in permanent tissue damage. Workers trapped in an area of heavy smoke may be asphyxiated. Where a burn escapes and causes a wildfire, severe worker injuries and fatalities may result. Wildfire and heavy smoke also may endanger members of the public in adjacent areas. In the extreme case, wildfire may trap and kill members of the public.

Long-Term Effects

Lower levels of smoke from prescribed burns may have a local, transitory effect on air quality. Sensitive members of the public may experience eye, throat, or lung irritation from these exposures. There is some risk that chronic, low-level exposure of workers or the public to smoke may lead to such health effects as emphysema, lung cancer or chronic respiratory disease.

Toxicity of Smoke Constituents

The various components of forest fire smoke are fairly well known, but the quantities produced vary considerably, depending on fuel moisture and fire temperature (Oregon Department of Environmental Quality 1987). The hazards include particulate matter, gases (carbon monoxide, carbon dioxide, and oxides of nitrogen), and chemicals that

may enter the lungs on the surface of particulate matter.

There are few studies which evaluate the toxicity of forest fire smoke. Almost all investigations of the toxicity of smoke particulate matter in human populations have been conducted with particulates associated with burning coal or fossil fuels (for example, automobile exhaust) where sulfur oxides and sulfates are the important constituents. However, these chemicals are not generated in a significant quantity by vegetation fires.

The vast majority of particulate matter generated by forest fires is thought to be in the fine particulate (FP) range: 90 percent is less than 2.5 microns in diameter (Oregon DEQ 1987b). These small particulates can be inhaled deeply into the lungs and deposited there.

Some components, such as many polycyclic aromatic hydrocarbons (PAH), are carcinogenic. Probably the most carcinogenic is benzo-a-pyrene (BaP), which has been demonstrated to increase in potency when mixed with carbon particulates (Dost 1986). Other components, such as the aldehydes, are acute irritants. These are most likely to affect forest workers who receive high exposures at the burn site.

In some cases, irritants unique to a certain plant may be released upon burning. The specific toxic agent in poison oak has been responsible for a large number of workers being out of work for long periods because of slow recovery.

Fire and Smoke Exposures

Fire Exposures

Worker exposures to fire depend on the number of prescribed burns and the acreage per burn. Worker exposures to wildfire depend on the number of prescribed burns that escape. Public exposures to fire depend on the number of escaped wildfires that are not properly controlled. Public exposures should be rare given the normal precautions used in prescribed burning.

Smoke Exposures

The gaseous components of smoke generally decompose or are diluted relatively quickly (Dost 1986). Other components, such as the aldehydes, may attach themselves to the particulates formed, and thus remain more concentrated and possibly protected from decomposition (Dost 1986). For these reasons, exposures to smoke have generally been estimated by measuring particulate concentrations in the air. This would not necessarily be a good estimate for people in close

contact with fire smoke, such as the workers.

Estimating the concentration of toxins in fire smoke involves determining drift, dilution, and breakdown of the various components. Knowledge of these factors and the ability to predict smoke exposures based on them are not well developed. Estimates of risk are based on the few data available on measured particulate concentrations.

While some smoke dispersion models do exist, there has been no generally accepted application of these models to smoke dispersion of forest fires. However, direct measurements of air concentrations of particulate matter have been made in communities located near areas of forest slash burns (Oregon Department of Environmental Quality 1986). Five locations were sampled in 1985, based upon proximity and wind direction (downwind) from areas with field and slash burning. Total suspended particulates (TSP) and particulate matter under 10 microns (PM-10) were measured (Oregon Department of Environmental Quality 1986). The highest 24-hour average TSP measurement was 83 micrograms per cubic meter (ug/m^3). The highest 24-hour average PM-10 measurement was 60 ug/m^3. These studies represent estimates of the maximum impact of slash burning on air quality over population centers.

Exposures for Forest Service workers managing prescribed fires or fighting wildfires would be much greater than these exposures. No direct measurements of worker exposures have been made, and it is not possible in this analysis to estimate with any degree of accuracy what smoke exposures would occur.

Quality of Information on Prescribed Burning Effects

Information on the likely effects of prescribed burning is poor. Most conclusions must be extrapolated from studies of air pollution from automobile emissions and other types of burning activities.

Risks from Prescribed Burning

Prescribed burning risks include the risk of wildfire, risk of physical injury to workers, and risk of chemical or particulate effects from smoke.

Risk of Wildfire

There may be numerous wildfires resulting from escaped prescribed burns, but they should be limited in size and should not present a serious risk to the public. All controlled burns require a burning prescription, which includes a description and discussion of

fuels, weather, and timing; how to burn; and safeguards. Personnel qualification standards and training requirements have been established for personnel involved in control burning. These requirements are directed toward reducing the risk of wildfire.

In the Pacific Northwest, fuel hazard abatement, coupled with increased efficiency in fire suppression, has reduced wildfire acreage (Tiedemann 1981). A significant proportion of the wildfires and acreage burned by wildfire on National Forests are attributable to escaped prescribed fires. From 1974 through 1984, escaped slash fires accounted for 7.5 percent of the wildfires and 27 percent of the burned acreage. Escaped slash fires averaged 34 acres in size.

Risk of Physical Injury to Workers

The risk of physical injury to workers is similar to that of manual methods. Workers on burn areas would be exposed to potential injury resulting from the manual treatments (brush piling, cutting) performed and the site conditions. Workers who manually light burn areas would be exposed to burning materials. There also could be a significant hazard associated with the mixing of gelled gasoline and diesel oil.

Data on typical casualty costs of controlled burning in Pacific Northwest forests, including 1 to 3 years of data from the Wenatchee National Forest and from two forest industries operating in northwest Oregon, suggest that one minor injury will occur for every 500 acres burned and one disabling injury will occur for every 7,500 acres burned. On three National Forests surveyed in the Pacific Southwest Region, there were 1,348 areas burned and only 15 personnel injuries, which is one for every 90 controlled burns.

Public safety would not be affected by any method of igniting burn areas. Most burning would occur in locations where the public either would not be present or would be highly visible to those doing the burning. Furthermore, those on or near a burning area would be well aware of impending activities because several hours of active preparation are required before ignition begins. Safety measures normally taken to protect firefighters participating in the prescribed burning also would protect the public.

Risk of Chemical or Particulate Effects from Smoke

It is not possible to accurately quantify human health risk from fire smoke. One of the major problems associated with the use of human epidemiology studies to evaluate the effects of fire smoke is the variation in the composition of the air pollutants studied. Most of these

studies have been carried out in urban areas with considerable particulate concentrations from automobile and industrial combustion of gasoline, diesel fuel, oil, and coal. All these sources include various components not found in fire smoke.

Air pollution has been clearly associated with mortality and morbidity. However, the health effects of air pollution caused by slash burning and wildfires have not been directly studied.

The highest level of TSP associated with slash burning observed in a populated area in Oregon was 83 ug/m^3. This level slightly exceeds the lowest estimated concentrations for intermediate particulates (IP) associated with chronic effects. The IP measurement is more restrictive than TSP, and thus the TSP overestimates the particulate concentration when compared to IP.

Thus, the highest level of particulate air pollution measured in a populated area in Oregon approximately equals the lowest level of particulate air pollution at which observed chronic effects have been reported. This level does not necessarily reflect a no-effect level. There is no evidence available to establish a no- effect threshold for fire smoke.

The confidence that no adverse human health effects will occur because of exposure to fire smoke at levels reaching population centers is uncertain. It is not possible at this time to quantify expected impacts. Observed particulate levels suggest that prescribed fire will not cause particulate levels to exceed the PM-10 standard (150 ug/m^3) proposed by the Environmental Protection Agency (EPA) (Oregon Department of Environmental Quality 1986).

Exposure of forest workers managing or fighting fires is likely to exceed levels with demonstrated health effects. Thus, the degree of confidence that no health effect would occur among these workers is very low.

An estimate of cancer risks due to PAH (polyaromatic hydrocarbons) from smoke inhalation was made by Dost (1986) using EPA's estimate of carcinogenic potency for BaP (a 0.0033 excess lifetime risk for cancer from continuous exposure to 1 ug/m^3) and assumed exposure levels for the public. His upper estimate of risk was in the range of one additional cancer per million people exposed (a one in 1 million individual cancer risk).

Conclusions

Workers are liable to experience injury and short-term effects from

fire and smoke. They are at particular risk from wildfires resulting when a prescribed burn escapes. The public is not liable to incur serious injury, although there is some indication that members of the public may experience long-term health effects from toxic constituents in fire smoke should they be exposed at relatively high levels that exceed state air quality standards. Workers are far more likely to experience cancer or chronic respiratory conditions from their exposures.

Biological Methods

Use of Biological Methods in Vegetation Management

Biological methods of vegetation management have included prolonged or forced grazing of cattle, goats, and sheep for conifer release and the selective introduction of parasitic insects for control of noxious weeds. In grazing for vegetation control, the water normally is provided, and livestock dispersal and movement is maintained through salting patterns and use of riders or herders. Attachment 4 to this risk summary profiles biological methods of vegetation management.

Adverse Health Effects of Biological Methods

Cattle or sheep are normally held in a plantation or confined area long enough to afford heavy utilization of feed and to generate a "release" effect in the crop trees. This combination of livestock numbers and duration of grazing may result in relatively high volumes of fecal matter deposited on the site. This factor, as well as the tendency for animals to concentrate in draw bottoms and adjacent to live water, creates a potential for fecal contamination of surface waters.

Exposures from Biological Methods

Members of the public who consume surface water downstream of biologically controlled sites may be exposed. Pathogens are not likely to contribute significantly to major municipal drinking water supplies, so larger populations are not likely to be at risk.

Quality of Information on Biological Method Health Effects

Little or no information exists on the spread of waterborne pathogens or the incidence of human illness that could be attributed to them from biological vegetation management.

Risks of Biological Methods

There is a remote possibility that fecal contamination of surface waters could result in the spread of waterborne diseases if animals were used to manage competing vegetation. It is not anticipated that

such contamination would impact health to any greater degree than normally occurs from wildlife in the forest. Stock tanks and methods to ensure animal movement and dispersal within the treatment area could be used as mitigation measures if necessary.

Conclusions

Human health effects are not likely to be caused by the use of biological methods. At present, there is insufficient evidence to conclude that waterborne disease is a human health effect of concern from the use of biological controls in this program. Downstream monitoring will be maintained in these projects where there is a question of potential human health effects.

Herbicides

Use of Herbicides in Vegetation Management

Herbicides are used in a variety of areas to control competing and unwanted vegetation. They most often are applied in mixtures with water or oil carriers, various adjuvants (wetting agents, sticking agents, stabilizers, enhancers, or thickeners), and dyes. The most frequently used methods for applying herbicides are helicopter and backpack applications for site preparation and release, trucks for right-of-way maintenance. Table 3-1 lists the 16 herbicides considered for use in Region 6, showing the specific vegetation management needs for which they are targeted. Attachment 5 to the risk summary describes the toxicology and quality of toxicity data for the 16 herbicides.

Adverse Health Effects of Herbicides

Conclusions about the toxic properties of herbicides are drawn from poisoning incidents, from laboratory studies of effects seen in human volunteers, from epidemiology studies, and from laboratory studies of effects seen in animals. Each of these types of information is associated with certain advantages and disadvantages, including uncertainties in predicting the effects of a chemical on an exposed individual.

Reports of poisoning most often indicate only the effects of very large doses, and the exact dose is seldom known. Studies on human volunteers, however, are confined to relatively small doses and are limited in duration. Epidemiology studies correlate disease observed in segments of the public with exposure to chemicals in the workplace or other areas. Results of epidemiology studies depend on data that is often gathered after the fact, and may be inaccurate. In many cases confounding factors, such as exposures to other chemicals or smoking, exist.

Table 3-1

Proposed Herbicide Program Use: Target Vegetation and Percent of Acreage Treated

Program Chemicals	Percent of Program Use	Treated Acreage
Herbicides		
2,4-D	Broadleaved weeds and herbaceous plants	38 %
Glyphosate	Broadleaved weeds and grasses	31
Picloram	Noxious weeds and shrubs	8
Triclopyr	Woody plants and broadleaved weeds	7
Dalapon	Annual and perennial grasses & sedges	Less than 5
Atrazine	Annual grasses	Less than 5
2,4-DP	Broadleaved weeds and herbaceous plants	Less than 5
Hexazinone	Most broadleaved weeds and grasses	Less than 5
Fosamine	Hardwood shrubs	Less than 5
Dicamba	Broadleaved weeds and brush	Less than 5
Asulam	Bracken fern, broadleaved weeds perennial grasses	Less than 5
Tebuthiuron	Woody range species	Less than 5
Diuron	Grasses and broadleaved weeds	Less than 5
Simazine	Annual grasses	Less than 5
Bromacil	Broadleaved weeds, grasses, shrubs	Less than 5
Amitrole	Annual & perennial grasses and herbaceous vegetation	Less than 5
Other Chemicals		
Diesel oil	Carrier for herbicides	—
Kerosene	Inert ingredient in formulations of 2,4-D and triclopyr	—

Laboratory animal studies are controlled methods of study. They examine effects under a range of high doses and various study durations. Uncertainty is involved in extrapolating the results of these studies to humans. Animal studies involve relatively few subjects and thus are not sensitive to small effects. How chemicals are absorbed, circulated, and metabolized varies by species, and animals differ from humans. High doses in animal studies are extrapolated to low doses in humans.

Poisoning incidents have shown that the 16 herbicides may cause severe, immediate reactions when received in high enough doses. However, such doses are rarely seen with these herbicides except in the cases of accidental or suicidal ingestion of concentrate. Even in these instances, the herbicides rarely have proven fatal. The herbicides may cause lower level immediate effects, such as nausea, dizziness, or reversible neuropathy. Longer term effects might include permanent nervous system damage; effects on reproductive success; damage to developing offspring; production of heritable mutations; damage to liver, kidneys, or other organs; damage to the function of the immune system; and cancer. These effects have been shown for a number of the 16 herbicides in laboratory animal studies, and there is suggestive evidence from epidemiology studies that these effects could occur; therefore, it is assumed here that there is a risk that they might occur at some dose levels in humans.

Toxic effects may be caused by the active ingredient in the herbicide formulation in a single dose, by a series of doses received over time (a cumulative dose), or by a combination of the herbicide active ingredient and another chemical (such as another herbicide, a carrier, or an inert used in the herbicide formulation).

Toxic Properties of the Individual Herbicides

Only a few of the 16 herbicides in the program have been examined in epidemiology or human volunteer studies, so judgments about risk rely most heavily on the results of studies in laboratory animals. The toxic properties of the 16 herbicides are summarized in three tables.

Table 3-2 categorizes health hazards for acute effects, general health effects, cancer and mutagenicity, and reproductive and developmental effects according to the compound evaluation system employed by the USDA Products Safety Inspection Service. This table is intended to show the maximum inherent toxic effects of a given chemical and indicates only the effects of very large doses. This table *does not* show effects expected from herbicides used in the Region's

vegetation management program. Actual and potential exposures from the program, even under worst case conditions, will result in doses very much smaller than the doses necessary for the health effects displayed in this table. Data on the specific effects are based on studies detailed in the three appendices on human health.

Table 3-3 describes effects reported in studies on systemic toxicity, cancer, and reproductive and developmental toxicity as well as testing for neurotoxicity and mammotoxicity. Summary statements indicate the principal effects reported with a dose sufficiently large to be toxic for each herbicide. This table is intended to show what the results could be if exposed to very large doses. This table does not show the effects expected from the Region's vegetation management program. Actual and potential exposures from herbicides used in the program, even under worst case conditions, will result in doses which are very much smaller than the doses necessary for the health effects displayed in this table. Data on the specific effects are based on studies detailed in the three appendices on human health.

The effects shown in Table 3-3 are summaries of effects reported at varying doses for tests conducted on laboratory animals and reported from human exposures. Not all effects were seen in all tests nor with all listed species. Any major effect seen has been noted regardless of the data quality and whether the results were of laboratory tests on rats, dogs, rabbits, or observations of humans. Testing protocols for necessary neurologic and immunologic effects have not been established by either the scientific community or the Environmental Protection Agency. In all but a few cases, existing data is inadequate. Sometimes neurologic or immunologic effects are noted in testing for other toxilogical endpoints. The reported effects are shown in the table.

Table 3-4 outlines the quality of the data on which the summary profiles in Tables 3-2 and 3-3 were based, and on which the overall assessment of risk in this document is based. Profiles of the toxicity and data quality of each herbicide are given in Attachment 5 to the appendix on Characterization and Management of Risk, jointly prepared by the Forest Service, LAI and the University of Washington. Detailed discussions of toxicity are given in Appendix D, compiled by LAI. Data quality is addressed in Appendix H, compiled by the University of Washington. The quality of data for each of six human health effects was categorized into one of four categories:

I Inadequate information available for evaluating toxicity. There were too few studies of sufficient quality to yield useful or reliable information.

M-I Some useable information exists for evaluating toxicity for the given health effect. There were some studies of marginal quality that provided useful information, but studies were inconsistent and some contained flaws. It is likely that new studies would change estimates of health effects.

M Marginal but useable information available for evaluating toxicity. There were studies of adequate quality and results did not vary greatly, but more information would increase reliability. Although new studies may change estimates of health effects, the results are considered moderately reliable.

A Adquate information is available. Studies are of sufficient quality and quantity that estimates of human health are considered reliable. New studies are unlikely to change estimates of human health effects.

Other Hazards

Inert Ingredients and Carriers

Inert and carrier ingredients are chemicals added to the active ingredient to facilitate the effective application of the pesticide. However, inert ingredients and carriers in certain formulations and under certain conditions have the potential to be a hazard to human health.

Assessing human health risks of inert and carrier ingredients is difficult because there is an extremely limited data base on human health effects and because inert ingredients are not stated on the label or made available to agencies or the public.

The Environmental Protection Agency (EPA) has identified about 1,200 inert ingredients that are used in registered pesticides. The EPA reviewed the existing human health data on inert ingredients (which include common carriers). The existing data include laboratory studies, epidemiological studies, and activity/structure relationships. The EPA categorized inert ingredients into four categories:

List 1: contains approximately 55 inert ingredients that have been shown to be carcinogens, developmental toxicants, neurotoxins, or potential ecological hazards. These ingredients are the highest priority for regulatory action.

List 2: Contains approximately 50 inert ingredients that have been given high priority for testing because the data is suggestive (but not conclusive) of possible adverse health effects or because they have structures similar to chemicals on List 1.

List 3: contains approximately 800 inert ingredients that are of lower priority for testing because no evidence from data or similarity of structure to chemicals on List 1 support a concern for toxicity or risk.

List 4: contains approximately 300 inert ingredients that are generally recognized as safe.

The Forest Service supplied the EPA with a list of all formulations of the 16 herbicides being considered for use in the Region. The EPA reported that the formulations of 14 of the herbicides proposed for use contain no ingredients on List 1 or List 2.

The esther formulations of triclopyr and the esther formulations of 2,4-D were reported to contain kerosene, which is classified as very slightly toxic (about 70 times less poisonous than 2,4-D) to laboratory rats. However, kerosene is a skin irritant. Kerosene was negative in five short-term assays for DNA damage and chromosome damage. Although it has not been shown to cause cancer in laboratory animals, it has a calculated cancer potency because it contains small amounts of such chemicals as benzene and BaP that are known to cause cancer. The calculated cancer potency of kerosene is about 6,000 times lower than the 2,4-D cancer potency.

Diesel oil is used as an herbicide carrier in tank mixtures. It is similar to kerosene in composition and contains benzene and BaP. Its toxicity is assumed to be about the same as kerosene. As indicated in Table 3-2, little information exists for categorizing the health hazards of kerosene or diesel oil.

The formulations proposed by the Forest Service are less acutely toxic than their active ingredient. However none of the herbicide formulations have undergone chronic toxicity testing, including cancer testing, or any reproductive, developmental, or mutagenicity testing. The inert ingredients in the proposed formulated products might cause cancer or other long-term health effects.

Given the little information that is available on each herbicide's formulation, the possibility that the formulated product is more toxic than the active ingredient cannot be discounted entirely. Neither can it be assumed to be true. For this reason, as well as others, very conservative assumptions that tend to overstate risk were made throughout the process of assessing risks contained in this EIS and the associated appendices. Based on information supplied by the EPA, this risk assessment assumes that synergism would not likely occur between the active and inert ingredients except in the formulations that use

kerosene. Given, as noted above, that the cancer potency of kerosene is about 6,000 times lower than the 2,4-D cancer potency, it would not add significantly to the potency of the 2,4-D formulation or mixtures.

Table 3-2

Summary of Potential Toxic Effects (Health Hazards) for 16 Herbicides

Herbicide	Acute (Poisoning)	Systemic (General Health)	Cancer or Mutagenic	Reproductive/ Developmental
2,4-D	Low	High	Low/Moderate Insufficient Info.	High
Glyphosate	Negligible	Low	Mod./Insuf. Info.	Moderate
Picloram	Negligible	Low/Mod.	Mod./Insuf. Info.	Low/Moderate
Triclopyr	Low	Moderate	Moderate	High
Dalapon	Negligible	Low	Neg./Insuf. Info.	Insuf. Info.
Atrazine	Low	Low/Mod.	Moderate	High
2,4-DP	Low	Moderate	Moderate	High
Hexazinone	Negligible	Low/Mod.	Low/Insuf. Info.	Low/Moderate
Fosamine	Negligible	Insuf. Info.	Insuf. Info.	Insuf. Info.
Dicamba	Low	Low	Low/Insuf. Info.	High
Asulam	Negligible	Low	Moderate	Moderate
Tebuthiuron	Low	Low	Neg./Insuf. Info.	Low/Moderate
Diuron	Negligible	Insuf. Info.	Insuf. Info.	Insuf. Info.
Simazine	Negligible	Low	Insuf. Info.	Moderate
Bromacil	Negligible	Low/Mod.	Moderate	Low
Amitrole	Negligible	Moderate	High	Insuf. Info.
Diesel Oil	Negligible	Insuf. Info.	Insuf. Info.	Insuf. Info.
Kerosene	Negligible	Insuf. Info.	Insuf. Info.	Insuf. Info.

This table categorizes health effects according to the compound evaluation system developed by the Food Safety Inspection Service. This table is intended to show the maximum inherent toxic effects for a given chemical and indicates only the effects of very large doses. This table does not show the effects expected from the Region's vegetation management program. Actual and potential exposure from herbicides used in the program, even under worst case conditions, will result in doses which are very much smaller than the doses necessary for the health effects displayed in this table. Data on the specific effects are based on studies detailed in the three appendices on human health.

Legend	Acute Toxicity: Oral LD50 (Mg/Kg of Body Weight)	Systemic Observed Damage to Vital Functions	Mutagenicity Carcinogenicity	Reproductive/ Developmental
High	25 or less	Irreversible	Mutagenic, confirmed carcinogenic (EPA Class A & B)	Adverse effects in rodents & nonrodents or conclusive in one species
Moderate	25-250	Serious and reversible	Weakly mutagenic, some evidence of carcinogenicity (EPA Class C)	Suspected adverse effects in one species
Low	250-1000	Transient and not permanent	Weakly mutagenic, negative cancer test	No adverse effects
Negligible	Greater than1000	None	Negative mutagenic and cancer test results (EPA Class D)	
Insufficient Information				

Table 3-3

Overview of Toxicity Information Available on the 16 Herbicides

Toxicity	Amitrole	Asulam	Atrazine
Systemic	Eye irritation, enlarged thyroid glands, reduced iodine uptake, decreased thyroid function, decreased body weight.	Gastrointestinal disorders, inflammation. Increased thyroid, heart, kidney, and body weights. Adrenal and pituitary gland changes in organ/body weight ratio. Fatty liver.	Eye and skin irritation. Decreased growth, food consumption. Decreased relative and absolute kidney and liver weights.
Cancer	Thyroid and liver tumors.	Questionable incidence of skin and subcutaneous undifferentiated sarcomas. Follicular thyroid hyperplasia. Possible pheochromocytoma.	Increase in female mammary adenocarcinomas and fibroadenomas. Increase in testicular interstitial tumors.
Reproductive	Hyperplasia of thyroid and atropic thymuses and spleens indicative of runting.	Decrease in number of live births. Reduced maternal weight gain.	Increased reabsorption. Reduced maternal weight gain.
Developmental	Abortions, structural changes, and decreased weight gain have been seen in some animal studies.	Minor abnormalities.	Increased visceral & skeletal variability. Weight loss. Fetal mortality.
Neurologic*	Insufficient information, no reported effects.	Vomiting and anorexia. Tremors, ataxia, hypoactivity. Learning impairment, alteration in EEG activity.	Insufficient information, no reported effects.
Immunologic*	Insufficient information, no reported effects.	Insufficient information, no reported effects.	Lymphopenia in rats.
Summary (General Toxicity)	Thyroid cancer.	Liver changes.	Body weight.

Toxicity	Bromacil	2,4-D	2,4-DP
Systemic	Gastrointestinal disorders. Decreased weight gain and growth. Changes in thyroid activity. Liver effects. CNS effects, impaired coordination.	Liver/kidney alterations. Decreased lymphocyte counts. Bleeding gums.	Blood enzyme effects, liver/kidney effects, decreased body weight.
Cancer	Liver tumors.	Increase in total malignant tumors. Brain tumors. Reticulum cell sarcoma. Liver, lung tumor increases. Lymphosarcomas, mammary tumors.	Pituitary and thyroid medullary tumors. Increase in rare malignant brain tumors.
Reproductive	Testicular abnormalities.	Decreased maternal survival. Increased fetal loss.	Decreased bulk. Reduced litter size. Pup mortality.
Developmental	Inadequate data.	Delayed ossification. Fetotoxicity. Skeletal abnormalities. Hydrocephaly.	Growth retardation. Skeletal malformations. Omphalocele.
Neurologic*	Insufficient information, no reported effects.	Histopathological lesions in CNS. Neuropathies. Memory impairment, polyneuritis. Brain lesions. Decreased nerve conduction velocity. EEG alterations. Myotonia.	Insufficient information, no reported effects.
Immunologic*	Insufficient information, no reported effects.	Altered immune function. In utero effects on lymphocytes. Antibody production suppressed.	Insufficient information, no reported effects.
Summary	Thyroid/liver changes.	Kidney/neurological.	Liver/clinical chemistry.

Toxicity	Dalapon	Dicamba	Diuron
Systemic	Kidney/liver effects.	Eye irritant. Skin rashes. Decreased food consumption and body weight. Liver changes, muscle cramps, dizziness, nausea, vomiting, voice loss, swelling of cervical glands.	Slight anemia, enlarged spleens, increased erythrogenic activity in bone marrow. Liver effects. Abnormal blood pigments. Weight loss. Nervous depression. Decreased respiration and heart rate. Weakness. Lethargy.
Cancer	None reported.	Considered negative, but some evidence of lymphoma and angiosarcomas suspected in 1 study.	Inadequate testing.
Reproduction	Maternal toxicity.	Maternal mortality. Decreased food consumption and body weight. Maternal ataxia.	Decreased maternal body weight.
Developmental	Decrease in maternal weight gain. Fetal resorption. Decreased pup weights.	Skeletal changes. Increased fetal resorptions. Reduced fetal body weights.	Fetal weight decrease. Wavy ribs. Delayed ossification. Sternoschisis.
Neurologic*	Insufficient information, no reported effects.	Sciatic nerve damage.	Insufficient information, no reported effects.
Immunologic*	Insufficient information, no reported effects.	No reported effects.	Increased spleen weight.
Summary	Kidney changes.	Body weight.	Effects on blood and liver.

Toxicity	Fosamine	Glyphosate	Hexazinone
Systemic	Increased heart, stomach, brain, kidney weights. Mild skin irritation. Possible changes in blood enzymes.	Decreased food consumption and body weight gain. Liver/kidney effects. Increased mortality. Decreased pituitary weight.	Lethargy. Irregular respiration. Eye irritation. Vomiting. Reduced weight gain. Liver changes. Decreased albumin. Increased alkaline phosphatase. Tremors, convulsions.
Cancer	No specific studies.	Testicular tumors. Renal tubular adenomas.	No oncogenic effects observed.
Reproductive	Inadequate data.	Maternal toxicity included inactivity, death, stomach hemorrhages, decreased weight gain.	Maternal decreased food consumption and weight gain.
Developmental	Hydronephosis (urine in kidney). Resorptions.	Delayed ossification. Structural malformation.	Abnormal skeletal development. Delayed ossification in extremities and extra ribs.
Neurologic*	Tremors and convulsions.	Insufficient information, no reported effects.	Insufficient information, no reported effects.
Immunologic*	Insufficient information, no reported effects.	Insufficient information, no reported effects.	Insufficient information, no reported effects.
Summary	Stomach weight.	Kidney changes. Liver weight.	Liver weight. Body weight.

Toxicity	Picloram	Simazine	Tebuthiuron
Systemic	Sensitizing reactions in humans. Increased liver weights. Altered liver cells. Mortality. Kidney lesions. Alterations in blood chemistry, body and organ weights.	Eye and skin irritant. Reduced erythrocyte and leukocyte counts. Elevated cholesterol and inorganic phosphate. Decreased body weight gain and food consumption. Decreased albumin; increased globulin, ketone levels. Urinary specific gravity.	Decreased growth. Hepatotoxic effect. Weight suppression. Increased thyroid and spleen weights. Pancreatic lesions. Increased liver and kidney-to-body weight ratios. Enzyme values increased.
Cancer	Benign adrenal, pituitary, liver, mammary and thyroid tumors. Small intestine adenocarcinomas. Increased neoplasms of spleen.	Inadequate testing.	No evidence.
Reproductive	Maternal toxicity. Reduced fertility at highest dose.	Tremors, abortion, decreased body weight gain and decreased food consumption.	Decreased body weight of weanling pups.
Developmental	Delayed bone ossification.	Reduced mean fetal weight. Increased skeletal variations. Late resorptions.	Inadequate data.
Neurologic*	Insufficient information, no reported effects.	Tremors. Labored breathing. Incoordination. Paralysis. Decreased brain weight. Hypoactivity. Muscular weakness. Convulsions. Ataxia.	Hyper-irritability. Tremors. Convulsions. Loss of righting reflex. Ataxia. Emesis.
Immunologic*	Insufficient information, no reported effects.	Insufficient information, no reported effects.	Insufficient information, no reported effects.
Summary	Liver weight.	Blood cell count.	Thyroid weight. Reduced growth.

Toxicity	Triclopyr
Systemic	Eye and skin irritant. Decreased weight gains, food consumption, absolute liver weights, growth rate. Liver and kidney effects. Gastrointestinal irritation. Increased mortality.
Cancer	Lung tumors. Mammary tumors.
Reproductive	Maternal decrease in body weight gain and food consumption.
Developmental	Maternal toxicity. Retarded ossification of skull bones. Fetotoxic effects. Increased resorptions.
Neurologic*	Insufficient information, no reported effects.
Immunologic*	Insufficient information, no reported effects.
Summary	Liver changes.

* This table describes effects reported in studies on systemic toxicity, cancer and reproductive toxicity, as well as testing for neurotoxicity and immunotoxicity. Summary statements indicate the principal effects reported with a dose sufficiently large to be toxic for each herbicide. This table is intended to show what the results could be if exposed to very large doses. This table does not show the effects expected from the Region's vegetation management program. Actual and potential exposures from herbicides used in the program, even under worst case conditions, will result in doses which are very much smaller than the doses necessary for the health effects displayed in this table. Data on the specific effects are based on studies detailed in the three appendices on human health.

The effects shown in this table are summaries of effects reported at varying doses for tests conducted on laboratory animals and reported from human exposures. Not all effects were seen in all tests, nor with all listed species. Any major effect seen has been noted regardless of the data quality and whether the results were laboratory tests on rats, dogs, rabbits or observations of humans.

Testing protocols for assessing neurologic and immunologic effects have not been established by either the scientific community or the Environmental Protection Agency. In all but a few cases, existing data is inadequate. Sometimes neurologic or immunologic effects are noted in testing for other toxilogical endpoints. The reported effects are shown in the table.

Table 3-4

Quality of Information on 16 Herbicides by Type of Toxicity

Chemical	Systemic	Cancer	Reproductive	Developmental	Neurologic	Immunologic
Amitrole	M	M	I	A	I	I
Asulam	M	A	M	M	I	I
Atrazine	M	M	M	M	M	I
Bromacil	M	M	M	M	I	I
2,4-D	A	M	A	M	A	M
2,4-DP	A	M	M	A	I	I
Dalapon	M	M	I	M	I	I
Dicamba	I	M	M	M	I	I
Diuron	I	I	I	M	I	I
Fosamine	M-I	I	I	I	I	I
Glyphosate	M-I	M	M	A	I	I
Hexazinone	M	A	M	M	I	I
Picloram	A	M	M	M	I	I
Simazine	M	I	M-I	A	I	I
Tebuthiuron	M-I	M	M	M-I	I	I
Triclopyr	M-I	M	M	A	I	I
Diesel Oil	I	M-I	I	M	I	I
Kerosene	I	I	I	M	I	I

Quality of Data:

I = Inadequate information available for evaluating toxicity. There were too few studies of sufficient quality to yield useful or reliable information.

M-I= Some useable information exists for evaluating toxicity for the given health effect. There were some studies of marginal quality that provided useful information, but studies were inconsistent and some contained flaws. It is likely that new studies would change estimates of health effects.

M = Marginal but useable information available for evaluating toxicity. There were studies of adequate quality and results did not vary greatly, but more information would increase reliability. Although new studies may change estimates of health effects, the results are considered moderately reliable.

A = Adequate information is available. Studies are of sufficient quality and quantity that estimates of human health are considered reliable. New studies are unlikely to change estimates of health effects.

Herbicide Exposure

Two human populations, workers and the general public, may be exposed during herbicide applications. Worker personnel, such as mixer-loaders and backpack sprayers, are directly involved in treatment operations. Members of the public, including forest visitors and nearby residents, may be exposed to herbicide drift, to vegetation with herbicide residues, and to accidental spraying. They also could eat food or drink water with herbicide residues. Figure 3-1 indicates examples of routes of exposure to herbicides. Table 3-5 lists the routes of exposure for which doses were estimated in the quantitative risk assessment.

The herbicide exposure analysis determined the extent of exposures and resultant doses to workers and the public from routine operations and accidents under the Forest Service's proposed program. Because no analysis could consider all the possible circumstances of herbicide spraying, scenarios were used that were simplified descriptions of spraying operations and potential routes of human exposure.

The scenario estimates were designed to indicate what could possibly happen under specified cicumstances. They do not reflect what will happen in the actual programs because worker restrictions and precautions should reduce worker exposures, and members of the public should not be exposed at all.

Four scenarios (helicopter, truck, backpack, and hand application) were used to estimate realistic worker doses in routine operations (routine-realistic scenarios). Four additional scenarios with the same methods of application were used to estimate the highest doses workers might get in routine operations (routine-worst case scenarios). The worker dose estimates were derived from actual worker field study data of 2,4-D doses found by urine analysis. These studies showed that inhalation exposure was a negligible contributor to a worker's total dose; therefore, no separate analysis of worker inhalation doses was conducted. Scenario dose estimates were adjusted for hours worked, application rate, and the different skin penetration rates of the other herbicides.

Three of the four routine-realistic scenarios (helicopter, truck, and backpack spraying) were used to estimate public doses. The same three routine-worst case scenarios were used to estimate the highest likely public doses. Because no studies of public exposure comparable to the worker studies were available, the public's doses were estimated by a mathematical accounting (modeling) of the movement and fate of

Figure 3-1 Routes of Exposure to Herbicides in Spraying Operations

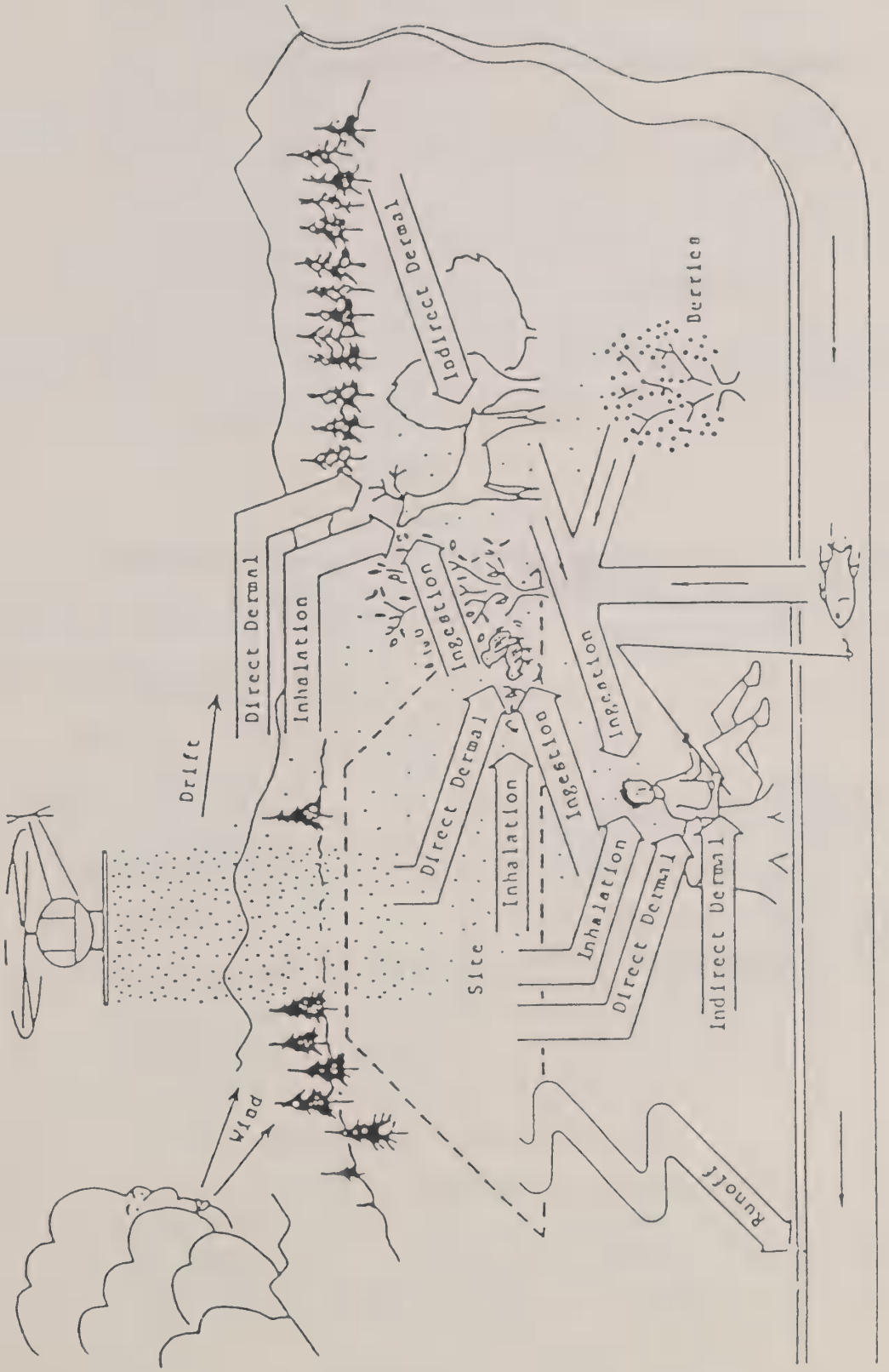


Table 3-5

Routes of Exposure Considered in This Risk Assessment

	Doses from Direct Exposure	Doses from Indirect Exposure
Routine		
Workers	Total dose (based on field studies)	Dermal dose from reentry to treated area (based on field data)
General Public	Dermal dose[a] from drift (based on modeling)	Dermal dose from vegetation contact in drift area and from consuming food with residues (based on modeling)
Accidental		
Spraying	Dermal dose[a] to member of public directly sprayed (based on modeling)	Worker vegetation contact dose from reentry to treated area immediately after spraying. Dose to member of public who walks through treated area and who eats directly sprayed food items[b] (based on modeling)
Spills	Worker dermal dose from spill of concentrate or mixture on skin (based on modeling)	Dose to member of public from drinking water contaminated by an herbicide spill (based on modeling)

[a] Inhalation is negligible based on field study data.

[b] Diet items include garden vegetables, berries, deer meat, game bird meat, and fish.

each herbicide in the area of spraying to estimate exposure levels on their skin, on their food, and in their drinking water. As in the case of workers, inhalation exposures of the public were considered negligible and were not estimated.

Dermal doses from the smaller herbicide spray droplets drifting offsite and depositing on a person's skin or from brushing up against vegetation with drift residues were estimated. Doses to a person drinking water with herbicide residues and eating berries, deer meat, game birds, garden vegetables, and fish containing herbicide residues also were calculated. It should be noted that these are one-time or once-per-year doses for the public that will be compared (by computing a margin of safety) to no-observed-effect levels (NOELs) derived from lifetime animal studies. This tends to greatly overestimate the risk from these rare events.

Estimated doses varied depending on the pesticide used, level of work, and which population (workers or the public) were considered. To put these doses into perspective, a person who takes a single vitamin C tablet of 250 mg each day and who weighs 70 kg (about 154 pounds) would be getting a dose of 3.6 mg/kg/day. If the person took a vitamin C tablet only once during his/her entire lifetime and lived 70 years, his/her average daily dose over those 70 years would be about 0.00015 mg/kg/day from that one tablet.

Realistic public doses ranged from 0.000001 mg/kg to 0.003 mg/kg. Worst case public doses in routine operations ranged from 0.000002 mg/kg to 0.25 mg/kg. Worker doses in routine operations ranged up to 0.6 mg/kg for backpack applicators for the realistic scenarios and up to 7.4 mg/kg for backpack applicators in the worst case scenarios. Doses to the public in accidents were as high as 4.4 mg/kg from a truck spill into a pond. Accidental doses to workers ranged up to 240 mg/kg for a spill of concentrate on their skin. Appendix D contains a full listing of doses for all scenarios.

Quality of the Scientific Data on Herbicides

To summarize, the quality of toxicity data for drawing inferences about possible human health effects are adequate for 2,4-D, 2,4-DP, and picloram. Studies are of sufficient quality and quantity that estimates of human health are considered reliable. New studies are unlikely to change estimates of health effects.

The overall quality of data for amitrole, asulam, atrazine, bromacil, dalapon, glyphosate, hexazinone, and simazine is judged to be marginal and has useable information for evaluating toxicity. There were

studies of adequate quality and results did not vary greatly, but more information would increase reliability. Although new studies may change estimates of health effects, the results are considered moderately reliable.

The quality of data for dicamba, tebuthiuron and triclopyr was judged to be marginal to inadequate. There were some studies of marginal quality that provided useful information, but studies were inconsistent and some contained flaws. It is likely that new studies would change estimates of health effects. Very cautious assumptions were made in characterizing risk.

Data was inadequate for diuron and fosamine. There were too few studies of sufficient information to yield useful or reliable information. More detailed information on the quality of data for all herbicides is shown in Table 3-4.

Risk of Herbicides

Risks to humans exposed to the 16 herbicides were quantified by comparing the scenario dose estimates with the doses from the toxicity tests on laboratory animals. Systemic effects were evaluated based on the lowest systemic NOEL. Reproductive effects were evaluated based on the lowest maternal, fetotoxic, or teratogenic NOEL.

For doses that are not likely to occur more than once, such as those received by workers spilling spray mix over their entire upper body, a dose estimate that exceeds the NOEL does not necessarily lead to the conclusion that there will be toxic effects. All the NOELs in this risk analysis are based on (or take into account) long-term exposure.

A worst case analysis of cancer risk was conducted for the herbicides for which there are clearly positive animal cancer studies (amitrole, asulam, atrazine, bromacil, and 2,4-DP) and for herbicides for which there is uncertainty or scientific controversy regarding their carcinogenic potential (2,4-D, glyphosate, and picloram). The risk of cancer is calculated for an individual by multiplying estimates of lifetime dose over a 70-year period by the herbicide's cancer potency. The risk of these herbicides causing mutations is qualitative rather than quantitative, with a statement of the probable risk based on the weight-of-evidence of mutagenicity assays and carcinogenicity tests.

Risk of General Systemic and Reproductive Effects

Risk to the Public

As indicated previously, we have explored the question of what could happen under unusual circumstances by developing hypotheti-

cal exposure scenarios. These scenarios do not represent risks that we actually expect to occur. Most members of the public would be at no risk at all because they are not in the immediate vicinity where herbicides are used. The only members of the public who are at risk are those who are actually exposed to herbicides as a result of being in an area where herbicides are being or have been recently applied, such as timber harvest units, or adjacent to roadsides where vegetation has been sprayed, or as the result of accidents.

Tables 3-6 and 3-7 summarize the results of the detailed analysis of risk to the public shown in full detail in Appendices D and H. Table 3-8 summarizes the most serious concerns raised by this part of the risk assessment and the actions that will be taken to address those concerns.

The four compounds in major use are 2,4-D, glyphosate, picloram, and triclopyr. Glyphosate and picloram present a low risk of systemic health effect under all scenarios of routine operations. Triclopyr and 2,4-D pose a moderate risk in these situations. Under accidental conditions, 2,4-D, picloram, and triclopyr have a high probability of effects on general health, while glyphosate poses a moderate risk. This type of physiological effect probably would not be detected by most individuals, but might be experienced as sickness by more sensitive members of the population.

Under routine operations, none of these four herbicides have a high potential for causing adverse effects to the reproductive system or to the developing fetus. 2,4-D, glyphosate, and triclopyr have a moderate potential for adverse effects. Under accidental conditions, 2,4-D, glyphosate, and triclopyr have a high potential for causing these effects, and picloram poses a moderate risk. The importance of taking adequate precautions to prevent members of the public from exposures from aerial applications of pesticides is confirmed by this analysis.

Of the 12 herbicides that have been used less extensively in the past, the Forest Service has decided to suspend the use of three: amitrole, fosamine, and diuron. Amitrole is suspended from use primarily because there is so much evidence that it causes cancer and because it appears to be able to negatively affect the developing fetus. Fosamine and diuron are suspended because there is no information available to evaluate cancer-causing potential in animals or humans for either compound. Reevaluation will occur when new information is developed.

Table 3-6

Estimates of Effects on General Health for Member of Public if Exposed to Herbicides Used in Routine Operations or from a Large Spill[a]

Herbicide	Probability of Receiving a Toxic Dose if Exposed During: Routine Operations	A Large Spill
2,4-D	Moderate	High
Glyphosate	Low	Moderate
Picloram	Low	High
Triclopyr	Moderate	High
Dalapon	Moderate	High
Atrazine	High	High
2,4-DP	Low	High
Hexazinone	Low	High
Fosamine	Insufficient Information	
Dicamba	Low	Moderate
Asulam	Low	Moderate
Tebuthiuron	Moderate	High
Diuron	Insufficient Information	
Simazine	Moderate	High
Bromacil	Low	High
Amitrole	High	High
Diesel oil	Insufficient Information	
Kerosene	Insufficient Information	

Based on criteria developed by the USDA Food Safety Inspection Service in the compound evaluation system (see USDA 1988). The categories for exposure and associated margins of safety are:

Probability of exposure to a toxic concentration	Calculated margin of safety
High	Less than 10
Moderate	Between 10 and 100
Low	Between 100 and 1,000
Negligible	Greater than 1,000

See Appendices D and H for complete information.

Table 3-7

Estimates of Effects on Reproductive System for Member of Public if Exposed to Herbicides Used in Routine Operations or from a Large Spill[a]

Herbicide	Probability of Receiving a Dose That May Cause Reproductive or Developmental Effects if Exposed During: Routine Operations	A Large Spill
2,4-D	Moderate	High
Glyphosate	Moderate	High
Picloram	Negligible	Moderate
Triclopyr	Moderate	High
Dalapon	Insufficient Information	
Atrazine	High	High
2, 4-DP	Low	High
Hexazinone	Low	Moderate
Fosamine	Insufficient Information	
Dicamba	Moderate	High
Asulam	Low	Moderate
Tebuthiuron	Moderate	High
Diuron	Insufficient Information	
Simazine	Moderate	High
Bromacil	Negligible	Moderate
Amitrole	Insufficient Information	
Diesel oil	Insufficient Information	
Kerosene	Insufficient Information	

Based on criteria developed by the USDA Food Safety Inspection Service in the compound evaluation system (see USDA 1988). The categories for exposure and associated margins of safety are:

Probability of exposure to a toxic concentration	Calculated margin of safety
High	Less than 10
Moderate	Between 10 and 100
Low	Between 100 and 1,000
Negligible	Greater than 1,000

See Appendices D and H for complete information.

Table 3-8

Summary of Concerns for the Public, Arising from the Herbicide Risk Assessment and Action That Will Be Taken to Address Those Concerns

Herbicide	Concern	Action
Amitrole	Strong evidence from animal studies that it causes cancer and could possibly affect fetal health; high risk under routine scenario for general health andreproductive and fetal health effects.	Suspend from use.
Fosamine	No information on cancer causing potential.	Suspend from use until information is developed.
Diuron	No information on cancer causing potential.	Suspend from use until information is developed.
Atrazine	Public Margins of Safety less than 100 in realistic aerial spraying scenarios.	Do not apply aerially.
2,4-D Dalapon, Diacamba, Glyphosate, Simazine, Tebuthiuron, Triclopyr	Public Margins of safety less than 100 in worst-case aerial spraying for range management scenarios.	Take all precautions to ensure public is not exposed in range management spraying (public notice,posting, avoid areas with residences).

Use of the other 12 herbicides in routine operations, presents high risks from atrazine and amitrole for systemic effects and from atrazine for reproductive effects. Dalapon, tebuthiuron, and simazine pose moderate risks of both systemic and reproductive effects, and dicamba presents a moderate risk of reproductive effects. The remaining herbicides pose low or negligible risks under routine operations. Accidental scenarios again show that the risk increases, especially for bromacil, hexazinone, amitrole, and 2,4-DP. For 2,4-DP, the increase in risk borders on the high category (also see Appendix D). Because there is an indication in a good animal model (the rabbit) for humans that 2,4-DP might cause birth defects, it will have restrictions on its use until further study shows the restrictions to be unwarranted. Once again the importance of accident prevention is emphasized.

The highest estimated dose to the public for most of the herbicides, major and minor use, comes in the scenario where someone drinks water from a pond, unaware that it has been seriously contaminated by herbicide concentrate from a truck accident (Appendix D). Consumption of water from this contaminated pond would present a sometimes moderate, usually high, risk for systemic and reproductive effects. Therefore, it is important to have an herbicide emergency spill notification system. The analysis shows that prompt restriction of access should prevent serious poisoning incidents.

Risk to Workers

Tables 3-9 and 3-10 summarize the results of the risk assessment for workers for general health, reproductive fitness, and/or fetal health if the worker is pregnant. All workers in these analyses were not wearing protective clothing. An analysis of risk with protective clothing was also undertaken and is shown in Appendix D which also shows in greater quantitative detail the risks summarized here.

Atrazine presents a significant risk of reproductive or developmental effects to backpack sprayers and aerial mixer/loaders. Use of atrazine, 2,4-D, and bromacil is also associated with significant risk to general health to backpack sprayers. In general, backpack spraying is associated with the greatest risk, followed by aerial mixer/loaders and hack and squirt workers. All other categories of workers (see Appendix D) have less risk according to this analysis.

It is true that the scenarios and doses estimated in the exposure assessment were designed to emphasize safety. Different scenarios with different assumptions could change the risk assessment. However, taken together with the analysis of risk with protective clothing, the risk assessment emphasizes the importance of attention to worker

Table 3-9

Probability of Receiving a Dose that May Cause Effects on General Health (Systemic Effects) of Workers Exposed to Herbicides During Routine Operations[a,b]

Herbicide	Aerial Mixer/Loader	Backpack Sprayer	R/O/W Mixer/Loader	Hack-&-Squirt
2,4-D	Moderate	High	Moderate	Moderate
Glyphosate	Low	Low	Negligible	N/A
Picloram	Negligible	Negligible	Negligible	Negligible
Triclopyr	Low	Moderate	Negligible	Low
Dalapon	Moderate	Moderate	Low	N/A
Atrazine	High	High	Moderate	N/A
2,4-DP	Negligible	Negligible	Negligible	Negligible
Hexazinone	Low	Moderate	Negligible	N/A
Fosamine	——————Insufficient Information——————			
Dicamba	Low	Low	Negligible	Low
Asulam	Low	Low	Negligible	N/A
Tebuthiuron	Low	Moderate	Negligible	N/A
Diuron	——————Insufficient Information——————			
Simazine	Moderate	Moderate	Low	N/A
Bromacil	N/A	High	Low	Low
Amitrole	Moderate	High	Low	Moderate
Diesel Oil[c]	——————Insufficient Information——————			
Kerosene[d]	——————Insufficient Information——————			

[a] Based on historical data of workers not wearing protective clothing.

[b] Based on criteria developed by the USDA Food Safety Inspection Service in the compound evaluation system (see USDA 1988). The categories for exposure and associated margins of safety are:

Probability of exposure to a toxic concentration	Calculated margin of safety
High	Less than 10
Moderate	Between 10 and 100
Low	Between 100 and 1,000
Negligible	Greater than 1,000

See Appendices D and H for complete information.

[c] Herbicide carrier

[d] Formulation inert ingredient

Table 3-10

Probability of Exposure to a Toxic Concentration for Reproductive or Developmental Effects in Workers During Routine Operations[a]

Herbicide	Aerial Mixer/Loader	Backpack Sprayer	R/O/W Mixer/Loader	Hack-&-Squirt
2,4-D	Low	Moderate	Low	Moderate
Glyphosate	Low	Moderate	Negligible	N/A
Picloram	Negligible	Negligible	Negligible	Negligible
Tricolopyr	Low	Moderate	Negligible	Low
Dalapon	———	Insufficient Information	———	
Atrazine	High	High	Moderate	N/A
2,4-DP	Negligible	Negligible	Negligible	Negligible
Hexazinone	Low	Low	Negligible	N/A
Fosamine	———	Insufficient Information	———	
Dicamba	Low	Moderate	Negligible	Moderate
Asulam	Low	Low	Negligible	N/A
Tebuthiuron	Low	Moderate	Negligible	N/A
Diuron	———	Insufficient Information	———	
Simazine	Moderate	Moderate	Low	N/A
Bromacil	N/A	Moderate	Low	Low
Amitrole	———	Insufficient Information	———	
Diesel Oil[c]	———	Insufficient Information	———	
Kerosene[d]	———	Insufficient Information	———	

[a] Based on historical data of workers not wearing protective clothing.

[b] Based on criteria developed by the USDA Food Safety Inspection Service in the compound evaluation system (see USDA 1988). The categories for exposure and associated margins of safety are:

Probability of exposure to a toxic concentration	Calculated margin of safety
High	Less than 10
Moderate	Between 10 and 100
Low	Between 100 and 1,000
Negligible	Greater than 1,000

See Appendices D and H for complete information.

[c] Herbicide carrier

[d] Formulation inert ingredient

safety training, use of protective clothing, emphasis on good personal hygienic habits in the workplace and in the field, and carefully defined procedures to minimize spills.

The risk from accidental spills of concentrated herbicides on the skin of unprotected workers is estimated in Appendix D. The estimated doses shown in Appendix D are usually greater than 1 percent and often greater than 10 percent of the LD_{50} and are likely to have serious harmful effects.

Cancer Risk

A worst case analysis of cancer risk was done only for those herbicides showing positive evidence (amitrole) or suggestive evidence (asulam, atrazine, bromacil, 2,4-D, 2,4-DP, glyphosate, and picloram) of causing tumor growth in the laboratory. For the remaining herbicides, there is either no evidence to suggest that they could cause cancer or there is not sufficient data to draw conclusions about their cancer causing potential.

Cancer Risk to the Public

Cancer risk for the general public was calculated for a single exposure, and also for 30 exposures over a lifetime. Of the eight chemicals, the greatest risks are for amitrole. In the highest exposure situation, the large aerial scenario, the maximum risk of cancer for a single exposure is less than 7 in 10 million, for a person eating vegetables from near the spray site. Even when several routes of exposure are added for the example nearby resident, the resulting risk for a single incident is still less than 2 in 1 million. The cumulative risk resulting from several exposures of this magnitude would be the sum of the risks for each exposure. If 30 routine-worst case exposures are experienced, the cumulative risk would be less than 3 in 10,000. Exposures in the routine-realistic cases lead to much lower risk. The risk of cancer resulting from doses from any of the routes of exposure in the typical aerial spraying scenario is less than 2 in 100,000, even for amitrole. Cancer risks for the other chemicals are far less. The risk for glyphosate is never greater than 2 in 1 billion. For 2,4-D, 2,4-DP, asulam, and picloram, none of the routes of exposure in any scenario results in a cancer risk greater than about 2 in 10 million, per exposure. The highest risk of cancer to the public from bromacil usage is from backpack spraying of large areas because bromacil is not aerially applied in the forests of Region 6. The risk from any route of exposure is less than 4 in 100 million.

Cancer Risk to Workers

Cancer risk to workers was calculated for an expected case assuming 5 years of employment in herbicide application, and an average number of days of spraying per year. The average number of exposure per lifetime was estimated to range from 30 to 70. The risk was calculated in the extreme cases assuming 30 years of employment and a total of 288 to 480 exposures. It is very unlikely that a worker would apply herbicides on the number of days assumed in the worst case. The risks for each herbicide were calculated assuming that only that herbicide was used. The highest risks for workers involve atrazine use. The lifetime cancer risk to a backpack sprayer using only atrazine is about 2 in 10,000 in the expected case. In the worst case the risk is greater than 1 in 1,000.

The risk is much less for the other chemicals. The highest risk for 2,4-D is about 1 in 100,000 for backpack spraying in the realistic case, and in the extreme case, the greatest risk is about 1 in 10,000. The risk is somewhat greater for amitrole: as high as 7 in 100,000 for the realistic backpack exposure. Workers using asulam in the extreme case have a lifetime cancer risk of less than 6 in 10,000 in all worker categories. Workers using bromacil have a risk of less than 8 in 10,000 in the worst case. The cancer risk from picloram or glyphosate use is even less for all worker categories. The risk in the expected case never exceeds 3 in 10 million.

Risk Of Other Health Effects

Heritable Mutations

As discussed previously, no human studies are available that associate any of the herbicides with heritable mutations. Furthermore, no risk assessments that quantify the probability of mutations from the herbicides are available in the literature or from EPA. While laboratory studies constitute the best available information on mutagenic potential, no acceptable mutagenicity test exist for some of the herbicides. For these herbicides, a worst case assumption is made that the herbicides have the potential to cause mutations in humans. In these cases the results of carcinogenicity tests or cancer risk assessments can be used to estimate the worst case risk for nonthreshold toxicity.

Using the results of carcinogenicity tests or cancer risk assessments is reasonable because mutagenicity and carcinogenicity both follow similar mechanistic steps for genetic toxicity. The basis for this assumption is that both mutagens and at least primary carcinogens react with DNA to form a mutation or DNA lesion affecting a particular gene or set of genes. The genetic lesions then require specific metabolic

processes to occur, or the cell must divide, to insert the lesion into the cell's genetic code. For these reasons, the quantitative risk of cancer provides a worst-case approximation to heritable mutations because cancer involves many types of cells, whereas heritable mutations involve only germinal (reproductive) cells.

Asulam and glyphosate tested negative for mutagenicity in all assays conducted, and thus can be considered to pose no mutagenic risk. Haxazinone and simazine were nonmutagenic in most assays conducted and were nononcogenic in all of the carcinogenicity tests performed; therefore, it can be assumed that their germ cell mutagenic risk is slight to negligible. Dicamba was nonmutagenic in most of the assays performed, and no oncogenicity was found in long-term studies. Because of the bulk of negative results, dicamba can be considered a mutagen in the worse case analysis, but the germ cell mutagenic hazard would be extremely limited. Bromacil tested positive in one of two oncogenic studies. The risk of heritable mutations from the chemical should be no greater that the estimates of cancer risk.

Atrazine tested positive for mutagenicity in 15 of 33 assays. The worst case assumption is that atrazine is mutagenic. However, many of the positive results were achieved through tests that may not be relevant to evaluating mutagenic risk in humans. Some positive results in rodents were also achieved, but these in vivo responses were only observed at levels greater than 1,500 mg/kg body weight. These are exceptionally high levels and suggest that the degree of germ cell risk would be lower than the risk for cancer and that the DNA change from low levels of atrazine would be minimal. The worst case estimate for atrazine mutagenic effects would be no greater than the risk of cancer.

Amitrole was nonmutagenic in 56 microbial gene mutation tests. The results of two tests that were positive are considered of questionable validity by EPA, and overall, it is considered to pose no potential for heritable mutations. The worst case estimate for amitrole mutagenic effects would be the risk of cancer.

For picloram and 2,4-D, studies have indicated positive and negative mutagenic potential. Most picloram tests were negative. About half the 2,4-D assays were positive or weakly positive. A number of comprehensive reviews of the 2,4-D mutagenic data have indicated that it does not pose significant risk of human gene mutations (U.S. Department of Agriculture 1984b). Based on a worst case estimate, the risk of heritable mutations from these chemicals would be no greater than the estimates of cancer risk.

Mutagenicity tests with 2,4-DP have shown mixed results. 2,4-DP was negative in 3 microbial assays and positive in 3 other assays; therefore, it may have limited genotoxic potential. Based on the limited test data presented in Section 3, one cannot presume mutagenic hazard, because no in vivo or mammalian assays have been conducted. However, the worst case assumption is that 2,4-DP is mutagenic and the mutagenic risk in the worst case would be no greater than the risk of cancer.

Risk of Synergistic Effects

It is possible that synergistic effects could occur as a result of exposure to two or more of the herbicides considered in this analysis. It is unlikely that synergistic adverse effects could result from exposure to more than one herbicide applied in separate projects. Herbicide residues are not expected to persist from one application to another. The 16 herbicides are known to be rapidly excreted from the body, and public exposures to the herbicides should be low (except for accidents) and should occur only very infrequently.

Simultaneous exposure to more than one chemical is likely in cases where those chemicals are combined in a single spray mixture. Although most vegetation control projects in the Region would involve only a single herbicide, some areas would be treated with a mixture of herbicides, but only mixtures that have been approved for use by the EPA. The EPA guidelines for assessing the risk from exposures to chemical mixtures (EPA 1986b) recommend using additivity models when little information exists on the toxicity of the mixture and when components of the mixture appear to induce the same toxic effect by the same mode of action. In these instances synergism is not anticipated to occur.

Effects on Sensitive Individuals

Based on the current state of knowledge, individual susceptibility to the toxic effects of the 16 herbicides cannot be specifically predicted. Safety factors traditionally have been used to account for variations in susceptibility among people. The margin-of-safety approach used in this risk assessment takes into account much of the variation in human response as discussed earlier by Calabrese (1985). The normal margin-of-safety of 100 for both types of variation is sufficient to ensure that most people will experience no toxic effects. However, unusually sensitive individuals may experience effects even when the margin-of-safety is equal to or greater than 100. For example, there have been a few cases of peripheral neuropathy among the thousands of people exposed over the years to 2,4-D. Sensitive individuals comprise only a

fraction of the population at large, and it is not likely that a sensitive individual would be among those few people who might be exposed to any of the Forest Service's applications.

Cumulative Effects

No one individual member of the public is likely to receive repeated exposures to any of the herbicides because of the remoteness of most treatment units, the widely spaced timing of repeated treatments, and the use of a variety of herbicides for different purposes. This risk assessment used the lowest NOELs found in chronic animal laboratory studies for comparison with estimated human doses. The risk analysis results showed that risks to the public from realistic treatment are high only for amitrole. Risks are low to negligible for the other 15 herbicides. Thus, members of the public could receive doses of these herbicides repeatedly over the years, and still not suffer toxic effects.

It is the goal of the Forest Service to prevent public exposures. Due to the precautions taken routinely in treatment operations, the likelihood of a member of the public receiving one dose is very low. The chance of receiving multiple doses is extremely low, making the likelihood of cumulative effects negligible.

Conclusions

Two of the herbicides evaluated for program use, diuron and fosamine, had insufficient data to support a toxicological analysis. They should be eliminated from the program. A third herbicide, amitrole, poses unacceptable cancer risks under normal exposures. It also should be eliminated from the program. The remaining herbicides can be used safely in the program with precautions and certain restrictions that are described in the next section on program risks.

Characterization and
Management of Risk

Application of Risk Assessment Program Risks

Application of Risk Assessment: Program Risks

Summary of Program Risks

The vegetation management program for Region 6 presents risks from the use of all management methods for workers and from the use of prescribed burning and herbicides for members of the public. Workers may be injured using hand tools or mechanical equipment. They may be injured, burned, or suffer the effects of smoke inhalation in prescribed burning. They may suffer acute or chronic health effects from herbicide exposures. The public may be affected by smoke from prescribed burns or may be affected by low level chronic exposures to herbicides. Their risks and those of workers will be mitigated through risk management procedures described in this section.

Comparative Risks

It is informative to view the human health risks of the Region 6 vegetation management program in the context of the human health risks to the general public and to workers in various occupations experienced every day in the Pacific Northwest. This section summarizes those background risks.

Background Health Risks in the Pacific Northwest

This section discusses background human health risks of injuries, cancer, and other diseases for people living in the Pacific Northwest. As is true for the U.S. population as a whole, people in the Pacific Northwest are exposed to risks from automobile accidents; contaminants in the air, water, and soil; chemicals in the diet; and many other injuries and diseases. Occupational risks may be different than those that face the general public, depending on the work environment. Some of these risks can be quantified, while lack of data allows only a qualitative description of others. Also, in some subject areas, information is available for the United States as a whole, but not specifically for the Pacific Northwest. In such cases, it is assumed that the U.S. data apply to conditions in the Pacific Northwest.

Sources of information for this section include detailed discussions by the Centers for Disease Control of the 10 leading work-related diseases and injuries (as determined by the National Institute for Occupational Safety and Health [NIOSH]) (Centers for Disease Control 1987), summaries of vital statistics (births and deaths) for Washington and Oregon, the National Research Council's **Regulating Pesticides in Food—the Delaney Paradox and Injury in America** and Calabrese

and Dorsey's **Healthy Living in an Unhealthy World.** Except for certain infectious, notifiable diseases, there is little statistical information on nonfatal conditions, including cancer, that either are cured or are not the primary cause of mortality.

Risks from Injuries

Injury Incidence

Seventy million Americans incur nonfatal injuries every year. Among those less than 45 years old, injuries are the leading cause of hospitalization (National Research Council 1985).

NIOSH estimates that about 10 million traumatic injuries occur annually to people at work in the United States (Centers for Disease Control 1987). Several chronic injuries also are directly linked to the type of work done. For example, vibration syndrome affects up to 90 percent of workers using chippers, grinders, chainsaws, jackhammers, or other handheld power tools, causing blanching and reduced sensitivity in the fingers (Centers for Disease Control 1987). Noise-induced hearing loss affects 17 percent of U.S. production workers who are exposed to noise levels of 80 decibels or more on a daily basis (Centers for Disease Control 1987).

Injury Mortality

Approximately 140,000 Americans die from injuries annually. Of the 94,072 deaths from unintentional injury in 1982, 47.5 percent were due to motor vehicle accidents, 12.8 percent to falls and jumps, 6.8 percent to drowning, 3.7 percent to poisoning, and the other 29.2 percent to a wide range of causes (National Research Council 1985). Injuries are the major cause of death among young adults and children. From the ages of 15 to 24, injuries cause almost 80 percent of the fatalities (National Research Council 1985).

Injuries cause about 10,000 occupational fatalities per year Some of the causes include highway motor vehicle accidents (34.1 percent in 1980 to 1981), falls (12.5 percent), industrial-vehicle or equipment accidents (11.4 percent), and fires (3.4 percent) Workers in the mining and quarrying industry had the highest rate of traumatic deaths, at 55 per 100,000 workers. Agriculture had a rate of 52 deaths per 100,000 workers, while trade had only 5 deaths per 100,000 workers (Centers for Disease Control 1987).

In Washington, 1985 data show that out of 34,475 total deaths, 830, or less than 3 percent, were accidental, nonvehicular fatalities. Of these, 252 were from falls, 104 from poisoning, 98 from drowning, 69 from fire, at least 64 from mechanical trauma, and the rest from vari-

ous other types of accidents. Over one-half occurred in the place of residence (Washington State Department of Social & Health Services 1986).

Table 4-1 indicates that deaths from falls, fires, and accidental poisoning are relatively rare in both States. Forestry-related deaths from any of these causes are exceptionally rare in either State.

Table 4-1

Mortality Rates and Causes of Death in the Pacific Northwest

Cause of Death	Number of Deaths (Mortality Rate) Oregon[a]		Washington[b]	
All causes	23,328	(877.2)[c]	34,475	(786.4)
Heart disease (rate)	7,788	(292.8)	11,713	(267.2)
Cancer (rate)	5,272	(198.2)	8,007	(182.6)
Cerebrovascular disease	1,926	(72.4)	2,709	(61.5)
Accidents	1,184	(44.5)	1,635	(37.3)
Motor vehicle	638	(24.0)	805	(18.4)
Falls	No Data		252	(5.7)
Fire	No Data		69	(1.6)
Poisoning	No Data		104	(2.4)
Respiratory disease	1,090	(41.0)	1,951	(44.5)
All other causes	6,068	(228.2)	8,460	(193.0)

Sources: Oregon Department of Human Resources, 1987; Washington State Department of Social and Health Services, 1985.

[a] 1986

[b] 1985

[c] all number in parentheses are rates per 100,000

Risk of Cancer

Cancer Incidence

Nationwide, the chance of developing some form of cancer during one's lifetime is about 1 in 4 (Calabrese and Dorsey 1984 and National Research Council 1987). The causes of cancer development are many, including occupational exposure to carcinogens, environmental contaminants, and substances in food. In the United States, one-third of

all cancers have been attributed to tobacco smoking (Chu and Kamely 1988). It is estimated that work-related cancers account for anywhere from 4 to 20 percent of all malignancies (Centers for Disease Control 1987); however, it is difficult to quantify the information because of such factors as long intervals of time between exposure and diagnosis, personal behavior patterns, job changes, exposure to other carcinogens, and difficulties in documentation.

The diet plays a significant role in cancer incidence, with different estimates holding it responsible for anywhere from 30 to 90 percent of all cancer in humans (National Research Council 1982). Pesticide residues in food contribute to the total cancer risk encountered. Based on the review of oncogenic pesticides in food by the National Research Council (National Research Council 1987), overall risks of cancer are increased over the background cancer risk of 0.25 (1 in 4 lifetime risk) by 0.001 in the United States as a whole. Most of this increased risk is attributable to a single group of compounds, the fungicides, with herbicides contributing 27.1 percent (of the 0.001 increased risk), which is equal to 0.000271 increased risk. Virtually all of the herbicide risk is because of a single compound, linuron, which makes up 96.1 percent of the herbicide risk and 26 percent of the total risk from pesticide residues in food (National Research Council 1987). Linuron is not included in the herbicides proposed for use in this program. Any of the herbicides used in the Region 6 program would not increase an individual's lifetime risk by more than 0.000001 as an upper-bound general public estimate based on the exposures estimated in the quantitative risk assessment. Many other substances contained in foods have also been linked to cancer. For example, eating 15 pounds of peanut butter in one year gives an estimated risk of liver cancer of one in 83,000, because of the presence of aflatoxin, a waste product of a fungus that grows on certain grains and nuts under moist conditions (Calabrese and Dorsey 1984).

Cancer Mortality

Based on the data in Table 4-1, cancer accounted for 22.6 percent of all 1986 Oregon fatalities and 23.2 percent of 1985 Washington fatalities. These figures are reflective of the national cancer mortality figures, in which cancer accounted for 22.1 percent of 1985 deaths in the United States (U.S. Bureau of the Census 1988).

Risk of Diseases other than Cancer

Disease Incidence

According to the Centers for Disease Control (CDC 1987), clear causal links have been established between certain occupations and

specific illnesses. For example, asbestosis among insulation and shipyard workers has been linked to their exposure to asbestos, and pneumoconiosis among coal miners has been linked to the inhalation of coal dust. Occupational exposures to some metals, dusts, and trace elements, as well as carbon monoxide, carbon disulfide, halogenated hydrocarbons, nitroglycerin, and nitrates, can result in an increased incidence of cardiovascular disease. Occupational exposure to lead and ionizing radiation may lead to reduced male fertility. Female laboratory and chemical workers show a higher rate of miscarriage than the general population. Neurotoxic disorders can arise from exposure to a wide range of chemicals, including such commonly used pesticides as 2,4-D, methyl bromide, and organochlorine insecticides. Dermatologic conditions, such as contact dermatitis, infection, trauma, cancer, vitiligo, urticaria, and chloracne, have a high rate of occurrence in the agricultural, forestry, and fishing industries, with 2,233 reported cases in 1984 and an incidence rate of 28.5 per 10,000 workers.

Disease Mortality

The mortality rates for Oregon (1986) and Washington (1985) are listed in Table 4-1. The leading causes of death are listed, along with numbers of deaths and rates per 100,000. The Oregon death rate slightly exceeds the national average of 870 per 100,000. The Washington rate is well below the national average. Heart disease is the principal cause of death in both States. Other significant disease-related deaths include cerebrovascular disease and respiratory disorders.

Comparison of Risks Among Alternatives

Each of the alternatives has a potential for impacts on the health of both forest workers and the general public. The risk of health impacts is much greater for forest workers because they are subjected to repeated and more direct exposure to risk factors. Health risks to the general public are liable to be experienced primarily through exposures to herbicides or smoke that have been transported away from a project area by wind or water.

For this analysis, risks and effects are estimated only for those activities directly associated with vegetation management. The comparison includes analysis of:

- Injuries from manual control of vegetation and brush;
- Injuries from the management and control of prescribed fires and wildfires;
- Health effects from exposure to smoke from prescribed fires and wildfires; and

- Health effects from exposure to herbicides used to manage vegetation.

The analysis does not include risks from activities that are incidental to vegetation management, such as transportation to job sites, and exposure to gasoline, exhaust fumes, and noise from engines (chain saws, tractors, helicopters, etc.). Although the risks from these exposures are real, there are far too many to cover them adequately in this analysis.

Table 4-2 shows the acres treated by each method for the eight alternatives. Comparisons that can be made by alternative using the acres treated in Table 4-2 include: (1) estimated number of worker accidents, and (2) risk to workers and public from herbicides and smoke.

Only forest workers, and not the public, are expected to be at risk from immediate injury from accidents. The risk of injury occurs primarily with the use of manual Table 4-2 methods or prescribed fire on rough terrain. The difference in risks between alternatives can simply be estimated from the total numbers of acres that receive manual or fire treatment in an alternative.

In this analysis, the number of injuries is estimated as follows. Minor injuries are expected to occur in prescribed fire treatments at the rate of two for every 1,000 acres treated. For manual methods, the estimated rate is 7.7 per 1,000 acres treated. For major injuries, the estimates are made only for prescribed fire treatments and are expected to occur at the rate of 0.13 per 1,000 acres treated.

Using the acre estimates from Table 4-2 and the accident rates discussed above, the risks from using manual methods and prescribed fire can be compared among alternatives (Table 4-3).

There are risks to both forest workers and the general public from exposure to herbicides and smoke. The difference in risks can be determined by comparing the numbers of acres in each alternative that would be treated with herbicides or would be burned by prescribed fire. The analysis does not try to estimate the actual number of people that might be affected by these exposures, because it would have to rely on too many disputed assumptions.

Table 4-2

Thousands of Acres Treated Yearly by Method and Alternative

	Alternative							
Method	A	B	C	D	E	F	G	H
Herbicide	0	59.9	0	26.8	47.9	64.1	76.6	51.5
Mechanical	184.6	167.2	44.9	111.6	166.9	201.0	155.6	162.0
Manual	99.0	77.8	17.7	57.8	95.1	80.1	85.7	79.7
Biological	14.8	.3	3.8	18.7	5.9	8.3	6.9	7.4
Fire	217.8	210.0	0	125.8	194.0	175.8	215.0	202.0
No treatment	24.4	19.9	15.6	23.8	24.1	36.1	21.53	31.8
Other	11.5	14.1	4.8	16.0	14.7	11.0	18.3	8.8
Total	552.1	553.0	86.8	380.5	548.6	566.4	579.6	543.2

Table 4-3

Anticipated Number of Worker Accidents Per Year
(from combined use of manual methods and prescribed burning, by alternative)

	Alternative							
	A	B	C	D	E	F	G	H
Minor injuries	1,250	1,053	189	741	1,152	1,004	1,126	1,017
Major injuries	30	29	3	19	27	25	30	26

Summary Description of Alternative Health Effects

The human health risks of the program alternatives are compared in Table 4-4 and summarized here. Alternative A prohibits the use of herbicides. It has a large decrease in acres treated with herbicides and smaller increases in prescribed fire, manual, mechanical, and biological methods. Some problems with adverse health effects from noxious weeds may occur. Under Alternative B, all methods of vegetation management are available consistent with the direction given in

applicable land management plans. Overall health risks to workers would be significant for minor injuries. Major injuries should be rare. Chronic health effects may occur from herbicides and smoke. In the "no action" Alternative C, there is a drastic reduction in the use of all methods. Some increases in risk because of noxious weeds or physical hindrances of unwanted vegetation are expected. All human health risks would be markedly reduced.

Alternative D stresses prevention and restricts efforts to control vegetation in areas where adverse effects of the vegetation are fairly certain. All methods other than biological are reduced. Health risks from noxious weeds and unwanted vegetation are not increased. Alternative D results in major reductions in both accidents and potential chronic toxicity.

Alternative E restricts all aerial spraying. All methods except manual and biological are reduced or remain the same. Health risks from noxious weeds and unwanted vegetation are not increased. Under Alternative E, there are likely to be moderate reductions in accidents and disabling injuries. Potential chronic toxicity probably will not change from those under current management Alternative B.

Alternative F restricts the use of prescribed fire methods. The use of fire is reduced considerably, while herbicide use is only slightly increased. Use of manual methods increases slightly, while mechanical methods increase considerably. Biological methods are slightly increased. Health risks from noxious weeds and unwanted vegetation are not increased. There would be a decrease in minor accidents and disabling injuries under this alternative. Some reduction in potential chronic health effects would be expected.

Alternative G calls for increased intensive vegetation management in the National Forests. Herbicide methods primarily will increase, with smaller increases in most other methods. Mechanical methods are moderately decreased. There would be an increase in minor injuries and disabling injuries under this alternative. There would be a moderate increase in potential chronic toxicity.

Preferred Alternative H is an attempt to maximize the effective use of the vegetation management tools while minimizing their human health impacts. Under the preferred alternative, the risk of accidents should remain about the same as under Alternative B, but the risk of herbicide chronic toxicity should be markedly reduced.

Table 4-4

Summary of Health Risks of Alternatives

Alternative	Manual Methods	Mechanical Methods	Prescribed Burning
A No herbicides	Highest risk alternative for injuries	Highest risk alternative for injuries	Highest risk alternative for injuries
B Current Management	633 major injuries 20 minor injuries	At lease 4 minor injuries workers & public	420 minor injuries smoke effects to
C No Action	Lowest effects	Lowest effects	Lowest effects
D Least Management	About half the injuries of Alternative B.	About half the injuries of Alternative B.	About half the injuries of Alternative B.
E No Aerial Spray	About the same as Alternative B.	About the same as Alternative B.	About the same as Alternative B.
F Reduced Fire	Slightly increased risk over Alternative B.	Significantly increased risk over Alternative B.	Reduced risk of injury and chronic effects.
G Increase Management	Slightly increased risk over Alternative B.	Slightly lower risk than Alternative B.	Risks about the same as Alternative B.
H Preferred	Slightly increased risk over Alternative B.	Slightly lower risk than Alternative B.	Slightly lower risk than Alternative B.

Biological Methods	Herbicides	Overall
High acreage could result in some risk	No Effects	Injury risk high; chronic risk very low.
Minimal risk of waterborne disease	Acute effects: risk in spills only; long term effects: risks to workers and public.	Injury risk high; chronic risk very low.
Lowest effects	No effects	Lowest program risk.
High acreage could result in some level of disease risk.	No effects	Lowest risks of all management alternatives except "no action".
Minimal risk	Reduced public risk. Worker risk somewhat reduced from B.	Lowered risk to public.
Minimal risk	Slight increase in risk to workers and public over Alternative B.	Some reduction in injuries; reduction in fire risk; slight increase in herbacide risk.
Minimal risk	Increased risks over Alternative B.	More injuries. Increased chronic health risks.
Minimal risk	Lower risk than Alt. B; 3 herbicides eliminated.	Reduced risks, especially to public.

Managing Risks

Expected Health Effects of the Preferred Alternative

Public Health Effects

The possible health effects to the public resulting from the preferred alternative for those individuals who receive some exposure may include some slight illness similar to light flu symptoms, including upset stomach, headache and muscle aches, a slight drop in body weight, fever, and possibly some minor changes in blood chemistry and liver function. If these effects occur at all in those few individuals in the public that have the highest exposures, the effects would be reversible within a few days and would be indistinguishable from common varieties of the flu. It is possible that some sensitive individuals would have more severe effects.

It is unlikely, however, that most members of the public would receive sufficient exposure to develop any of the above-mentioned effects. However, those members of the public that are highly sensitive to chemical substances in their environment because of preexisting disease or because of allergy should be advised of herbicide treatments in their area so they can take precautionary measures to avoid any potential exposure.

Worker Health Effects

The same types of effects would be seen in workers who have increased exposure. The difference between workers and the public is that the workers have a much higher potential exposure to the herbicides than the public. Therefore, the effects seen in the workers could be more severe than those seen in the few members of the public who might have any effects. Workers could experience more pronounced flu-like symptoms, loss of weight, changes in blood chemistry and liver and possibly kidney effects. There is also the possibility with 2,4-D of some peripheral neurologic effects, such as numbness in the fingers. These effects would be reversible in most instances. Workers who display any of the above effects should be reassigned to activities that do not involve exposure to herbicides. It should be noted that for the worst case scenarios for workers, high exposures and more severe effects may be anticipated. In worst case scenarios these effects may not be reversible and may even be life threatening; thus protective clothing and special work practices should be instituted to avoid accidents.

Decisions About Herbicide Use

Under the preferred alternative, the health risks to workers and the public will be managed to ensure that no one is subject to risks greater than background risks (above one in 1 million for cancer), and no one

is subject to chronic health effects based on a margin-of-safety (MOS) less than 100. This objective is met in three steps.

First, no herbicide will be used that (a) has insufficient data to determine its program risks with a reasonable degree of assurance, (b) presents cancer risks to workers and the public that cannot be limited to one in 1 million by restricting the types of applications in which it is used, or (c) gives an MOS less than 100 that cannot be mitigated by managing conditions of use.

Second, those herbicides shown to be possibly carcinogenic in laboratory animal studies will be managed to minimize exposures to workers and the public to the level where their risk does not exceed one in 1 million based on the upper 95-percent confidence limit of the estimated cancer potency of that herbicide. Herbicides shown to have possible systemic or reproductive effects on workers or the public also would be restricted. This second group may be further broken down into those herbicides that require special precautions only for workers and those requiring mitigation to reduce public exposures also.

Third, those herbicides 1) with MOS's all greater than 100 in all routine exposures and 2) not suspected of being carcinogenic or whose cancer potency is sufficiently low that normal safety precautions and mitigation measures will ensure that exposure levels are low and that their risks are no greater than one in 1 million will not be restricted beyond those measures identified on the pesticide labels.

The 16 herbicides are listed in the three groups as follows.

Group 1. Group 1 will not be used in Region 6:

- Diuron (insufficient data);
- Fosamine (insufficient data); and
- Amitrole (high cancer potency).

Group 2. Group 2 has special precautions to limit risk to acceptable levels. The mitigation measures used to limit worker risk to acceptable levels would include such precautions to minimize exposures as use of nonabsorbent gloves, long-sleeved shirts, eye protection, and face shields. Workers using these chemicals in these types of applications would be required to wear this protective gear, and their compliance with the requirement would be verified in regular worker monitoring.

- **Subgroup A.** Subgroup A of Group 2 has herbicides (atrazine, bromacil, dalapon, and simazine) that need to be restricted for

workers. Backpack application with these herbicides will only be made on low-growing vegetation (the target vegetation is grasses and other herbaceous weeds). The exposure for backpack application is extrapolated from a 2,4-D study (Lavy et al., 1984), in which workers sprayed brush 5 to 15 feet in height. In applying such grass killers as atrazine, workers would not raise the spray wand above arm height. This means that the predicted exposures and risks for backpack applicators are probably greatly exaggerated. Nevertheless, worker exposure will be reduced by verified use of protective clothing and reduced exposure periods and mandatory cleanup or sanitation measures.

- **2,4-D.** Restrict hack & squirt applicators using 2,4-D to wear protective clothing.
- **Subgroup B.** Subgroup B of Group 2 includes the herbicide atrazine that needs to be restricted for the public. It will not be used in aerial applications.

Group 3. Group 3 has no restrictions on use, normal safety procedures are sufficient to insure acceptable risk.

- **Subgroup A**. Subgroup A of Group 3 contains weak carcinogens, such as picloram (cancer risk below one in 1 million in all cases).
- **Subgroup B**. Subgroup B of Group 3 has noncarcinogens: dicamba, hexazinone, tebuthiuron, and triclopyr.

Mitigation Measures

Table 4-5 summarizes the human health effects of concern and risk management decisions made to mitigate the potential effects. In addition to the specific mitigation measures identified in Table 4-5, mitigation measures were developed as an integral part of developing this EIS to reduce, avoid, or minimize potentially adverse impacts on the environment that might result from vegetation management activities.

The mitigating measures were developed using Federal laws and regulations; the intent of State resource laws; existing direction found in the Forest Service Manuals and Handbooks; land and resource management plans; resource management experience; and research findings.

Several mitigating measures cover all vegetation management

activities, regardless of method. Others apply to a particular method. These mitigating measures are tied to methods. If a method is used in an alternative, the mitigating measures associated with that method will be followed.

What follows is a summary of the mitigation measures that apply to vegetation management. (For more information, refer to "Mitigation Measures and Vegetation Management Methods" in Chapter II of the Draft Environmental Impact Statement. Additional information on the effectiveness and impacts of mitigation measures is in Chapter IV, Environmental Consequences.)

Before using any method of vegetation management, Forest Service personnel will be required to: conduct an environmental analysis, including scoping, as required in Forest Service Manual 1950, for each proposed project; prepare a human health risk management plan for each project; and provide training and quality control at Regional, National Forest, and District Offices. The mitigation measures that apply to all silvicultural vegetation management require a documented prescription, prepared or approved by a certified silviculturist and a site-specific diagnosis that meets Forest Service Silvicultural Practices Handbook standards (2409.17) and treatment needs (2409.26c).

Each project proposal will include a human health risk management plan. The size and complexity of each vegetation management project, along with the specific methods and techniques employed, will define the content of the project risk management plan. The design of every project, however, will evaluate the health risks for workers and the public and make specific provisions for the management of risks. As appropriate, this may include such elements as:

a) Project Risk Plan. This includes the identification of personal protective equipment, special orientation and training needs, hazard identification, first aid training or supplies, and safety meeting schedule. The basic reference is Forest Service Handbook 6709.11 (Health and Safety Code Handbook). Medical support facilities, as well as accident investigation and reporting responsibilities, will be identified.

b) Environmental Monitoring Plan. This includes water quality monitoring procedures and standards, and identifies appropriate requirements for notification of adjacent landowners or forest visitors, and record-keeping needs.

c) Spill Incident Response Plan. This is for projects involving move-

ment or handling of hazardous materials. The plan will identify responsibilities, notification procedures, and containment or cleanup measures.

d) Prescribed Burning Plan. This includes identification of preburn monitoring of fuel moisture and weather conditions, the burning prescription, assessment of risk of fire escape, coordination needs, and special smoke management needs.

e) Herbicide Application Plan. This identifies organization and support needs, calibration procedures, equipment inspection procedures, communications needs, search and rescue procedures, batching procedures, public contact procedures, posting and signing needs, and herbicide accountability standards. Special precautions for applicators, mixers, and loaders also will be identified. This plan is not applicable to Alternatives A and C.

Biological Methods

When using livestock to control vegetation, the Forest Service will notify affected water users and ensure strict control of livestock near riparian areas. The release of insects to control specific vegetation requires coordination with State and Federal agencies. Site analysis will explore the seeding of compatible plants and the use of genetically superior seedlings, natural seedlings, and advance regeneration as ways to prevent or minimize the need for future vegetation management.

Manual Methods

When using workers with handtools and power tools to treat vegetation, safety risks will be analyzed and incorporated into the human health risk management plan.

Mechanical Methods

When mechanical methods of treating unwanted vegetation and logging residues are used, tractors will not be used on steep slopes, on highly compactible soils, on erodible soils in municipal watersheds, or during conditions with high risks to soils. Buffers of vegetation will be left along streams, lakes, and wetlands to minimize sedimentation. Slash will not be piled in stream floodplains.

Use of Herbicides

If herbicides are used, there will be strict adherence to EPA label instructions for the herbicide applicable State and Federal laws, and site-specific mitigation measures. In addition, these specific measures will be implemented: downstream water users who have the potential

of experiencing adverse human health effects from project operations and adjacent landowners will be notified of planned use of herbicides; precautions against accidental leaks or spills will be taken; mixtures will be prepared and equipment will be cleaned in areas outside sensitive watersheds, where groundwater will not be contaminated; spray droplet size will be optimized to minimize drift; specific buffers will be left along streams, rivers, lakes, and wetlands; pilot vehicles will be used when transporting mixed herbicides; and monitoring will be done to ensure effectiveness of mitigation measures during spray operations.

Herbicide use will be in compliance with the Forest Service Pesticide Use Manual (FSM 2150). Forest Service Handbook standards will be followed, specifically 2109.11 for planning projects; 2109.12 for storing, handling, and transporting herbicides and for spill prevention, cleanup, and disposal requirements; 2109.13 for defining worker training and experience requirements; and 6709.11 for identifying worker safety requirements. Individual National Forests will provide detailed guidance for large projects. Applicator training, testing, and licensing will be required to ensure knowledge of herbicide uses, risks, and safety. Herbicide safety data sheets will be posted at storage facilities and in vehicles and made available to workers.

Prescribed Fire

For the use of prescribed fire, extreme care will be taken to avoid excessive consumption of litter and duff; reduce fuel consumption on steep and erodible slopes, leaving unburned borders of vegetation along streams; protect air quality, following all State and local regulations; avoid intrusion of smoke into State-identified sensitive areas; use the best available technology to reduce smoke; (in Oregon) comply with Oregon State Implementation Plan prohibitions; and (in Washington) comply with the Washington State Smoke Management Plan and Implementation Plan.

Monitoring

This section indicates the types of monitoring that are considered part of the overall effort to manage program risks. They are employed to minimize the chances of human exposure or to minimize the chances that exposures that do occur will lead to adverse health effects.

Management Practices

Management practices will be monitored by supervisory personnel at the operations site. Adherence to safety practices and specific restrictions will be documented in the project report.

Accidents/Injuries

All worker accidents are tracked in a daily log on each forest, and

the type of work being performed is recorded. Type of outpatient or inpatient care is recorded, as well as the time lost in work and cost of medical care.

Application

Herbicide and fire projects will be closely monitored for appropriate weather conditions. Speed and direction of wind in relation to the spray or burn site are critical to a proper operation. In herbicide applications, card crews monitor the accuracy of applications by determining the location and level of spray droplets on specially prepared cards placed downwind of the site. Monitoring will be planned as an integral part of the overall vegetation management project. Monitoring will be conducted as described in the Region 6 Water Quality Monitoring Guide for Pesticide Detection (R6-WS-040-1980). Monitoring of a spray operation will be conducted to determine whether mitigation measures are (1) being observed, (2) effective in maintaining water quality, and (3) in compliance with State water quality standards.

Exposures

Exposure monitoring will be conducted for at least 10 percent of the projects where workers are involved in herbicide operations. Monitoring can include patches on clothing or urinalysis. This monitoring will be conducted more frequently where chemicals are used that have shown low MOS's in specific applications. This is the case for 2,4-D in backpack applications.

Environment

Environmental monitoring will be conducted for soil and water as determined by site specific project analysis of the site before application to establish a baseline level, immediately after application, and at intervals afterwards.

Health

Worker health will be monitored and documented by a screening interview conducted by the project leader before herbicide applications, and at monthly intervals during the spray season. Follow-up medical examinations will be conducted when there is indication of associated health problems.

Public health is monitored by communication with the local public health service for indications of effects that might be related to Forest Service operations, such as reports of bronchial irritations in areas near prescribed burns.

Table 4-5

Human Health Effects and Mitigating Measures

Human Health Effects	Mitigating Measures
Insufficient information on reproductive or developmental effects for dalapon.	Public: Eliminate the use of dalapon in roadside vegetation management or other uses where members of the public could be exposed to dalapon either through routine operations or accidents. Workers: Do not use female Forest Service or contract workers in any dalapon applications.
High probability for the public of receiving a toxic dose if exposed to amitrole during routine operations. A probable human carcinogen, high cancer potency.	Eliminate amitrole from the program. Mitigation measures may not be consistently effective in managing the risks.
Moderate to high probability of the public receiving toxic doses from a large accidental spill for most herbicides.	Constantly monitor location and condition of all tankers or trucks carrying herbicides for Forest Service operations. Prepare emergency spill plans including public notification for all projects as part of the project risk management plans.
Moderate probability of the public receiving a toxic dose of 2,4-D, dalapon, dicamba, glyphosate, simazine, tebuthiuron or triclopyr if exposed in a large aerial range management spraying project.	Take all precautions to ensure the public is not in the vicinity of the sprayed area including posting public notices. Avoid treatment of areas with nearby residences.
High probability of the public receiving a toxic dose of atrazine from a realistic aerial spraying.	Do not apply atrazine aerially.
Moderate probability of backpack applicators and hack-and-squirt applicators receiving a dose that may cause reproductive or developmental effects in operations for 2,4-D, glyphosate, dicamba, tebuthiuron, triclopyr, simazine, and bromacil. High probability of their receiving a dose that may cause these effects for atrazine.	Do not use contract or Forest Service female workers in backpack or hack-and-squirt applications of any of the listed herbicides.

Moderate-to-high probability of mixer/loader workers receiving a dose of atrazine or simazine that may cause reproductive or developmental effects.	Do not use female workers as mixers or loaders in atrazine or simazine applications.
Public health effects from exposure to smoke from prescribed burning.	Follow Oregon and Washington State smoke management plans.
Moderate or high probability of workers receiving a toxic dose if herbicides are spilled on skin during: 2,4-D aerial, backpack and hack and squirt operations; Triclopyr aerial, backpack and hack and squirt; Dalapon backpack; Atrazine aerial and backpack; Simazine aerial and backpack; Bromacil: backpack (high) ROW and hack and squirt; and Amitrole aerial and backpack.	Require wearing of protective clothing for all Forest Service and contract mixer loaders, backpack applicators, and all hack and squirt applicators. Require written certification that protective clothing was worn and the names of workers involved for both Forest Service and contract workers. Conduct field exposure sampling on 10% of projects for which workers may have moderate probability of receiving a toxic dose.

Any potential adverse human health effects.	Worker Exposure Monitoring: For all contract and Forest Service workers involved in pesticide application programs, keep a written record of names and jobs of individuals involved, chemical used, acreage treated, use of protective clothing and equipment, duration of exposure, and method of application. Institute a regional database for tracking the information on an individual's involvement in herbicide programs. Public Exposure Monitoring: Where a concern exists for public exposures, develop an exposure monitoring plan for the project which is appropriate to the concern, monitoring methods used, and the site. Monitoring may include water monitoring, monitoring of drift with spray cards, or taking soil or plant samples. Risk Management: For each vegetation management project develop a risk management plan that includes a project risk plan, environmental monitoring plan, spill incidence response plan, prescribed buring plan, and herbicide application plan.
Unknown health effects of brown and burning (using herbicides as a desiccant to facilitate prescribed burning).	Do not prescribe burn any areas within one year of treatment with pesticides.

Insufficient information to assess human health effects of kerosene and diesel oil.	Diesel oil will not be used in herbicide applications except as an adjunct (not to exceed 5% o the spray mixture). Kerosene will not be used in herbicide applications except as an inert ingredient in the esther formulation of triclopyr. A long history of using diesel oil and kerosene (in a variety of operations such as slash burning, fueling equipment and as a cleaning solvent) has not revealed acute human health effects. However, there is insufficient information available to asess long-term health effects. Care will be taken to avoid skin contact with diesel oil and kerosene. If contact does occur, affected skin areas should be promptly washed with soap and water, and soaked clothing will be changed.
Mixtures	All contract and Forest Service workers directly involved in applying herbicide mixtures must wear protective clothing, and follow the most stringent mitigation measures of any constituent.
Variable or unknown health effects of inert ingredients in herbicide formulations.	Use herbicide formulations that are known to contain only inerts on EPA inerts lists 3 and 4 (inerts of lower priority for testing and inerts generally recognized as safe). See Appendix J. For formulations that contain inerts on lists 1 or 2 (inerts of high priority for testing and inerts that have been shown to be hazardous), use formulations only when inerts are known and human health risks have been fully assessed according to the standards established in this EIS and incorporated in NEPA decision-making.

Effects on hyper-sensitive individuals.	Public notification for all applications that requests that people who know or suspect that they are hypersensitive contact the Forest Service to determine appropriate risk management measures. Institute a self-screening program to identify and remove sensitive individuals from herbicide projects. Both contract and Forest Service Workers that have knowledge of hypersensitivity not to be used on a project, on-the-job, or workers who display symptoms that may indicate hypersensitivity will not be used on application projects.
Cumulative herbicide cancer risk over a lifetime.	Monitor contract or Forest Service workers to identify those whose cumulative exposures due to herbicides lead to cancer risks that approach 1 in 1,000,000 (background risk) and remove those individuals from application projects.
Diuron: Limited data suggest that Diuron has relatively high toxicity and high exposure for backpack application. In addition, there is insufficient information for full toxicologic evaluation.	Eliminate diuron from the Region's program.
Fosamine: There is insufficient information for conducting a full toxicologic evaluation.	Eliminate fosamine from the Region's program.

Characterization and
Management of Risk

Attachments

Attachments

Attachment 1

Manual Methods Profile

Manual methods utilize hand labor to remove competing vegetation or noxious weeds or to modify the immediate environment so favorable conditions exist for desired plants.

Scalping during planting is one of the most commonly used manual methods. A small area is cleared with a handtool to remove potentially competing vegetaton in the vicinity of the planted tree. Mulching (with paper, plastic, or other materials placed on the ground surrounding the tree to prevent moisture loss) or the growth of species that compete with the target species are rarely practiced.

Power saws are commonly used to achieve release objectives. Competing brush is cut, allowing the crop tree more space and resources to grow. Use of power saws for release has increased since suspension of herbicide use in the Region. Hand pulling of weeds or small competing seedlings and girdling (removing a band of bark from around the stem) are occasionally done for conifer release. Hand pulling is frequently used at recreation and administrative facilities, tree nurseries, and occasionally along roadsides, where noxious weeds commonly invade.

As in all methods, timing of operations is critical. For example, the ability of brush to resprout partly depends on when it was cut, and the effectiveness of pulling depends on the timing of germination.

The advantage of hand methods is their specificity and low impact on the soil surface. Particular species can be targeted. In riparian areas and sites with sensitive plants, hand methods ensure that only target species are treated.

The major disadvantages of manual methods are their lower production rates, higher costs, and, in the case of release, resprouting. Because the root system remains intact, except after hand pulling, the plant retains its vigor and can quickly reoccupy the site.

Manual methods are extensively covered in the Reforestation Handbook (2409.26b) and the Timber Stand Improvement Handbook (2409.26c).

Attachment 2

Mechanical Methods Profile

Mechanical methods of vegetation management involve the use of machines to remove or reduce the cover of competing or unwanted plant species.

Crawler tractors or low ground pressure tractors outfitted with various types of blades or mowing attachments are the most commonly used techniques. Site preparation is most often accomplished using various types of blades to remove plants, their roots, and, sometimes, part of the top layers of soils. The technique is named for the extent of the activity. Preparing spots is called scalping; plowing a strip is called furrowing or contouring. In some cases, most of the area is prepared.

Tractors also are used to pile unmerchantable material that may present a fire hazard or create difficult conditions for reforestation. Tractors with attached discs or chains also are used to remove unwanted vegetation for range improvement. Graders, tractors, and other machines use attached brush cutters for roadside brush control.

Cable systems also are used to yard unmerchantable material that may present a fire hazard or create an obstacle to tree planting.

As with all other methods, the timing of application can affect the success and efficiency of the operation when using mechanical methods. Application is usually timed to avoid sprouting and high soil moisture content.

The advantages of mechanical methods are the low costs and high efficiency (in many cases, the plant, roots and all, is removed).

The intense disturbance of the site is the major disadvantage of mechanical methods, particularly during site preparation. Most techniques are nonselective and remove nontarget plants. There are slope and topographic limitations, except with cable systems, and there is usually some resprouting if the whole plant is not removed.

Mechanical methods, tools, and techniques are extensively covered in the U.S. Forest Service Reforestation and Timber Stand Improvement Handbooks (2409.26b and 2409.26c, respectively).

Attachment 3 Prescribed Burning Profile

Prescribed burning is the controlled use of fire under predetermined conditions to achieve specific, preplanned land management objectives. Prescribed burning, as a vegetation management method, has advantages. In the right situation, it can be environmentally correct and very cost-effective.

Like the other vegetation management methods, it does have disadvantages. First, fire is not very selective. The effects of fire on a particular organism are not always predictable, as they depend on fire severity and residence time, as well as the particular species' heat-fire tolerance. How long the effect lasts depends on each species' vigor, capacity for sprouting, and heat tolerance of its seeds.

If too much fuel is consumed, the soil can be damaged and water quality may be affected. The smoke from prescribed fires can degrade air quality and a prescribed fire may escape, become a wildfire, and do considerable damage to the environment as well as to manmade improvements.

There are three techniques of prescribed burning: broadcast burning; pile burning; and underburning. Broadcast burning is the burning of natural and activity-generated forest residues, scattered (more or less uniformly) over a clearcut. The most commonly used devices for igniting broadcast burns are handheld drip torches and helicopter. Helitorches are used when there is a need to ignite an area rapidly or when it is unsafe to light by hand. Rapid ignition makes it possible to burn at higher fuel moistures, thereby reducing the amount of smoke produced.

Mechanical pretreatment is often done in combination with broadcast burning. For example, brush or saplings may be cut prior to burning. Logging residues may be crushed to reduce fire intensity and rate of spread.

Broadcast burning, depending on the amount of fuel that must be removed, can be done during the spring, summer, and fall. This levels the workload and lessens the impact on air quality. Pile burning of forest residues is done in conjunction with "PUM" (piling of unmerchantable material) and "YUM" (yarding of unmerchantable material) to concentrate slash into piles or windrows. PUM is done manually or with a tracked or rubber-tired tractor; YUM is done using skidders or cable-logging machinery.

Generally, piles and windrows are burned in the fall after snowfall or rain to minimize the risk of escaped fires. The most commonly used

devices for igniting piles are handheld drip torches and alumagel packets. Alumagel is a gasoline-thickening agent. The jelled gasoline is put into plastic bags, placed inside the piled slash, and ignited electronially from a remote location.

Fire severity in pile burns is typically very high; soil under the burning pile is often sterilized by the high soil temperatures caused by the relatively long residence of the fire.

Underburning is burning beneath a stand of trees to reduce woody debris, set back unwanted vegetation, or encourage the growth of desirable forage and browse plant species. The most commonly used device for igniting an underburn is the handheld drip torch. Underburning is done only when fuels are barely dry enough for the fire to spread properly. Low air temperatures and moderate air movement are prescribed to dissipate the convective that would otherwise damage the crowns of the overstory.

Attachment 4

Biological Methods Profile

Generally, biological methods include the use of animals or insects to control vegetation. Grazing animals and leaf-eating insects are the most commonly used methods. There are, however, a number of less commonly used methods. Seeding of desired species, genetic adaptation, or use of natural and advance regeneration can be used successfully in certain situations. Other techniques involving biological herbicides, allelopathy, and pathogens are still experimental. Prolonged or forced grazing of domestic livestock (cattle and sheep) can be used to control the composition or amount of competing vegetation. This differs from the typical grazing program in that vegetation control, rather than animal weight gain or forage utilization, is the primary objective.

Livestock use can be considered when palatable or preferred species are a significant component of the vegetation and when an area large enough to support the herd or band is available for management. Careful coordination with range and wildlife habitat management goals is normally required.

Grazing can have several advantages. In the proper mix of brush, weeds, and grasses, grazing can effectively control the vigor of undesirable vegetation. Grazing can be cost-effective and may often be done in conjunction with existing range permits. On some nutrient-deficient sites, the animals can be beneficial because they convert vegetation directly into an available source of nitrogen.

Several disadvantages also must be recognized. Timely project administration and experienced herders or riders are needed to control the duration and intensity of use. This is particularly true with sheep movement and bedding. Conifer seedlings can be susceptible to browsing or trampling damage, especially during the spring season. Livestock must be strictly controlled within riparian areas or on soils subject to compaction in order to prevent damage to soil and water resources. Water sources, the extent of the forage, the quality or nutritional value, access, proper fencing, and control all can be limiting factors.

Experience has shown that willing operators are not plentiful. To obtain a release effect in conifer seedlings or significantly reduce undesirable vegetation, livestock must be held in an area much longer than normal. Forced grazing such as this can adversely affect animal weights and marketability—a serious problem for many stockmen.

Livestock have been effectively used to control competing vegetation in the rangeland rehabilitation programs on the National Forests of eastern Washington and Oregon. Sheep and cattle also have been used effectively for conifer plantation maintenance. Specific examples of successful livestock use on an operational basis can be found on the Fremont and Siuslaw National Forests.

The selective release of insects has been used to weaken or kill specific target plants in certain noxious weed situations. These biological control efforts require close coordination with several state agencies and county weed control programs, as well as Federal agencies, including the USDA Agricultural Research Service. Insect releases can be effective when the population of target plants is large enough to support the insects or nematodes, and adequate numbers of insects can be obtained through USDA Biological Control Laboratories.

Insect adults and larvae can damage noxious weeds by feeding on seeds, girdling roots, and forming galls. Efforts are normally made to harvest the insects for redistribution purposes. Selective release programs have a history of success in local situations. A great deal of research effort has been directed toward this technique in recent years.

The disadvantages of this biological vegetation management technique are the intensive monitoring efforts required and the difficulty of obtaining insects. While the introduction of host-specific insects is carefully studied and planned in advance, there is always the potential risk of disrupting natural ecosystems.

Some examples of host-specific insects successfully used in the Pacific Northwest to control target vegetation include the cinnabar moth (tansy ragwort); fleabeetles and midges (leafy spurge); seedhead weevils (yellow starthistle); root and stem boring weevils (Canada thistle and Scotch broom); and seedhead flies (diffuse knapweed).

Seeding with desired species is a preventive technique used on newly disturbed sites, such as roadsides and other rights-of-ways. Early seeding of low-growing grass and bush species, often accompanied by fertilization, may inhibit later invasion of the site by taller shrubs and trees. Once a stable plant community is established, the site becomes self-maintaining. More research is needed in a variety of site conditions to determine which species are successful under what conditions.

Through the Regional genetics program, the technique of genetic adaptation is being explored. Trees with the potential for fast, early growth are selected to be used as a seed source. The use of stock developed from these seeds may limit the need for conifer release in some situations. Thus far, the program has been limited to a few commercially important species. However, as results are evaluated, more species may become available and adapted for site-specific needs.

Taking advantage of natural seedlings and advance regeneration (established trees from the previous stand) can reduce, in some cases, the need for competition control. Natural seedlings go through a rigorous natural selection process and are uniquely and specifically adapted to the site. There usually are a number of different species present, adding to the diversity and increasing the chances for survival of a health stand. In many cases, they grow faster than the planted trees.

Using advance regeneration has the same advantages as using naturals, but their older age and large size can give them a significant advantage over the competition. However, advance regeneration stands can be diseased, suppressed, or damaged and do not always represent a positive opportunity.

Several biological techniques show promise in the experimental stage, but are not yet operationally proven in forestry. These include: 1) biological herbicides—naturally occurring microbial or viral agents similar to those which have proven effective in insect control and agriculture; and 2) pathogens and allelopathy—use of introduced pathogens and chemicals produced by plants to repel or inhibit competitors.

consistent with site and management requirements. Removing selected age classes may not allow the competitors to gain dominance on a site because many brush species require full sunlight for optimum growth. The remaining crop trees expand to take advantage of the newly available resources left after harvest.

To minimize soil disturbance and damage to the residual stand in an uneven age management regime, the terrain must be gentle. Otherwise, long-term damage caused by multiple entries would far outweigh the benefits to vegetation control. The Region and National Forests have standards and guidelines dealing with the selection of harvest systems.

Attachment 5 Toxic Properties of the Individual Herbicides

The primary data base on the toxic properties of the 16 herbicides being considered by the vegetation management program is based on laboratory animal studies. However, three of the herbicides have been examined in epidemiology studies or studies with human volunteers. This information will supplement the animal data for those herbicides. The following discussion briefly summarizes what is known about the toxicity of each herbicide and what data are missing. Appendices D and H contain a detailed discussion of each herbicide's toxicology. Appendix H also contains an examination of the quality of the toxicology studies and a discussion of epidemiology studies. With few exceptions, no data on neurotoxicity or immunotoxicity exist for the herbicides. Based on historical use patterns up to 1982, the Forest Service expects to rely most heavily on four pesticides: 2,4-D, glyphosate, picloram, and triclopyr. Together, these four have accounted for approximately 84 percent of the pesticide use in the past. The remaining 12 herbicides each have been applied to 5 percent or less of the total acreage in Region 6. They are discussed in approximate historical order of use.

2,4-D

2,4-D, a phenoxy acetic acid, is the most widely and commonly used herbicide by the Forest Service for the selective control of broadleaved weeds and woody shrubs. The toxicology of 2,4-dichlorophenoxy acetic acid has been reviewed by, among others, the U.S. National Academy of Sciences (1977), Hayes (1982), the World Health Organization (1984), the U.S. Environmental Protection Agency (1986), the U.S. Department of Agriculture (Newton and Dost 1981; USDA 1984; Shipp et al. 1986), the California Department of Food and Agriculture (1986), and the Canadian Centre for Toxicology (1987).

Acute Toxicity

The acute oral LD50 in laboratory animals ranges from 300 to 1,000 mg/kg. It can therefore be classified as a "moderately to very" toxic compound (Klaassen 1986). Adverse effects seen in acute studies include excessive thirst, loss of appetite, loss of weight, depression, roughness of coat, tremors, muscular weakness particularly in the hind quarters, rapid breathing, and salivation (USEPA 1984). 2,4-D can cause contact dermatitis in humans from occupational exposure (Poland et al. 1971). When dermal contact continues, nausea, vomiting, muscular weakness, and diarrhea have been reported, indicating absorption. However, according to Mullison (1981), ingestion of 8 mg/kg/day for 3 weeks or intravenous injections of 800 to 960 mg over 32 days or 21 days caused no measurable adverse effects in human test subjects. Injection of 3,600 mg (equivalent to 51.4 mg/kg/day) caused stupor, incoordination, weak reflexes, and urinary incontinence, all of which returned to normal in 24 hours.

General Systemic Toxicity

There is adequate testing in this area. Long-term animal studies have reported some effects on kidney function at low doses. The NOEL is less than 1 mg/kg/day.

2,4-D has been tested in several different laboratory animal species, including mice, rats, guinea pigs, rabbits, and monkeys. Substantial disagreements in the results of subchronic and chronic studies perhaps reflect the use of different formulations (NAS 1977). Signs of toxicity have included loss of appetite, loss of weight, muscular weakness, gastrointestinal disturbance, depressed growth, and increased liver weights (WHO 1984). While no-observed-effect levels (NOEL's) in rats administered 2,4-D acid by the oral route have ranged from 62.5 mg/kg/day in a 2-year study to 15 mg/kg/day in a 113-day study, in a recent 90-day feeding study in Fischer 344 rats, alterations in renal histopathology were observed at the lowest dose tested, 1 mg/kg/day (Shipp et al. 1986; EPA 1984). The EPA has used this lowest effect level (LEL) in their most recent risk assessments of systemic toxicity from 2,4-D exposure.

Reproductive Effects

There is adequate testing in this area. Animal reproductive studies have demonstrated fetotoxicity as well as other effects. The NOEL is 5 mg/kg/day.

Studies of small populations have produce mixed positive and negative results. More detailed information is presented in Appendix H of the EIS.

The lowest NOEL for a multigeneration reproductive study is 5 mg/kg/day, the lowest dose tested. Fetotoxicity was reported at 20 mg/kg/day, the next highest dose tested.

Developmental Effects

There is marginally adequate testing in this area. Animal studies have demonstrated developmental effects, including some minor malformations. Based upon delayed ossification, a LOEL of 12.5 mg/kg/day was observed.

Two rat developmental studies have found 25 mg/kg/day to be the NOEL because of fetotoxicity at higher doses (50 mg/kg/day and above) (EPA 1985; CDFA 1986; NAS 1977; Schwetz 1971), although some reviewers have concluded that in each of the two developmental studies, the lowest doses (8 and 12.5 mg/kg/day) were the more appropriate NOELS due to fetotoxicity at the higher dose of 25 mg/kg (Shipp et al. 1986).

Neurotoxic Effects

There is adequate data for a qualitative assessment. There is moderate evidence for neurotoxicity associated with 2,4-D exposure. This is based on mixed animal data and human data. The human data include information from poisonings and other acute exposures, as well as occupational exposures. Cases of loss of feeling in the hands and feet and contact dermatitis have been reported from occupational exposure to 2,4-D. No NOEL has been calculated.

Three cases of peripheral neuropathy have been reported in humans following dermal exposure to 2,4-D. This condition may be partially or totally reversible, depending on the dose level and the individual exposed. In one patient, only partial recovery was reported, even after 3 years of treatment (Goldstein et al. 1959).

Immunotoxic Effects

There is marginally adequate data for a qualitative assessment. There is only weak evidence of immunotoxicity associated with 2,4-D exposure based only on animal data.

Oncogenicity

There is marginally adequate testing in this area. Animal studies have shown mixed or questionable results. Based on mixed tumor (including neoplasms of the liver and lung, reticulum cell sarcoma, and benign tumors), the upper 95-percent confidence limit of the cancer potency was estimated as 0.005 per mg/kg/day.

Oncogenicity studies on rats, dogs, and mice have been reported in the literature using various esters of 2,4-D. According to the World Health Organization (1984) and the Canadian Centre for Toxicology (1987), the available evidence is either inadequate or insufficient to conclude that 2,4-D is an animal carcinogen. Most recently, a 2-year mouse feeding study reported a statistically significant increase in brain tumors over controls (CDFA 1986; Canadian Centre for Toxicology 1987). One review of this study concluded that the kind and number of tumors and the background evidence of oncogenicity within the study did not support the conclusion that the tumors were caused by the exposure to 2,4-D (Canadian Centre for Toxicology 1987).

A number of human epidemiology studies of phenoxy herbicides have been conducted, with some focusing more specifically upon 2,4-D. These studies have presented mixed results for a number of tumor sites. Positive studies have been reported for soft tissue sarcomas, Hodgkin's Lymphoma, Non-Hodgkin's Lymphoma, stomach cancer, and lung cancer. A more complete discussion of these studies is included in Appendix H of the EIS.

Genotoxicity studies on 2,4-D have included in vitro studies on bacteria, yeasts, and cultured mammalian cells. In vivo genotoxicity studies have been conducted on rats, mice, hamsters, and Drosophila. In addition, some limited studies have been carried out in humans exposed occupationally to 2,4-D. Overall, the often conflicting pattern of responses observed in both in vitro and in vivo tests indicates to expert reviewers that 2,4-D is either not genotoxic (Canadian Centre for Toxicology 1987), a weak mutagen without significance as an environmental mutagenic hazard (Newton and Dost, 1981) or not a potent mutagen (WHO 1984).

GLYPHOSATE

Glyphosate, a glycine compound, is a broad spectrum herbicide used to control herbaceous vegetation as well as shrubs.

Acute Toxicity

The acute oral LD50 of technical glyphosate in rats ranges from 3,930 to 4,750 mg/kg (EPA 1986), which suggests that glyphosate is a "moderately toxic" compound (Klaassen, 1986). The Roundup formulation containing a surfactant appears to be a potential skin irritant, based on rabbit studies. However, experiments on the skin of human subjects have been negative for skin irritation (USDA 1984).

General Systemic Toxicity

There is marginally adequate testing in this area, but the dose at which effects are seen in animal studies varies widely. A questionable NOEL, based on reduced pituitary weight, is 20 mg/kg/day. Most other NOELs are considerably higher.

Subchronic dog and rat studies have shown no significant abnormalities up to 2,000 ppm (USDA 1984); however, these studies were among those done by IBT that have been invalidated by EPA (1986). A chronic rat study found no toxic effects up to 31 mg/kg/day, the highest dose tested. A chronic mouse study reported increased hepatocyte necrosis at 30,000 ppm, resulting in a NOEL of 5,000 ppm, the next lowest dose tested. Preliminary analysis by the EPA (1986) suggests that in a recently completed dog feeding study, decreased pituitary weights at 100 mg/kg had results in the lowest NOEL of any study, 20 mg/kg/day. However, the CDFA (1986) reported "no significant adverse effects" at any dose in their analysis of the same study. In summary, animal studies have yet to clearly demonstrate adverse effects up to doses of 750 mg/kg/day. The EPA has used 31 mg/kg/day from the rat chronic study for its risk assessment process.

Reproductive Effects

There is only marginally adequate testing in this area. The NOEL is 10 mg/kg/day.

The EPA (1986) reports that a three-generation reproduction study with rats showed kidney effects (focal tubular dilation) in the pups at a dose of 30 mg/kg, resulting in a NOEL of 10 mg/kg/day, the next lowest does tested. However, CDFA (1986) considered that the kidney effects are probably not "significant adverse effects." The EPA (1986) used 10 mg/kg.day as a reproductive/developmental NOEL for risk assessment.

Developmental Effects

There is adequate testing in this area. A conservative NOEL (based on effects not clearly related to exposure) is 75 mg/kg/day. The next lowest NOEL is 1,000 mg/kg/day.

Two developmental studies, one using rats and the second using rabbits, found no treatment-related developmental effects from glyphosate at doses of 300 mg/kg/day and higher (EPA 1986).

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Animal studies have demonstrated only questionable excesses of cancers at high dose levels. However, glyphosate was assumed to pose a cancer risk in this assessment. Based on kidney tumors in mice, the upper 95-percent confidence limit of the cancer potency was estimated as 0.00003 per mg/kg/day.

Both previously described rat and mouse chronic assays have been judged as providing no evidence that glyphosate is an animal carcinogen. Kidney tumors at the highest dose tested (30,000 ppm) in the mouse assay have been judged not to provide sufficient evidence for oncogenicity (EPA 1987). Glyphosate was negative in all short-term assays conducted, including a CHO/HGPRT assay, a DNA repair assay, an in vivo bone marrow assay, a dominant lethal assay in mice, host-mediated assays in mice and rats, two Ames assays, and a rec assay.

PICLORAM

Picloram, a picolonic acid, is used for the control of woody plants and broadleaved weeds.

Acute Toxicity

On the basis that the acute LD50 in rats is between 2,633 and 4,000 mg/kg, picloram can be considered a "moderately toxic" compound (Klaassen 1986). Most formulations of picloram are not irritating to the skin. However, the mixture of 2,4-D and the triisopropanalamine salt of picloram may produce sensitizing reactions in humans (EPA 1987).

General Systemic Toxicity

There is adequate testing in this area. Picloram seems to be primarily a liver toxin. A NOEL based on increased liver weight is 7 mg/kg/day.

Based on available short-term and chronic studies with rats, mice, and dogs, picloram seems to be primarily a liver toxicant. The lowest NOEL from a laboratory animal study is 7 mg/kg/day. Increased liver weights in male beagles (but not in females) were observed at 35 mg/kg/day. Seven mg/kg/day was the next lowest dose tested (USDA, 1984).

Reproductive Effects

There is only marginally adequate testing in this area. The NOEL is 50 mg/kg/day.

According to the EPA (1984), a three-generation rat reproductive study had a NOEL of 50 mg/kg/day because of reduced fertility at 150 mg/kg/day, the next highest dose tested. In a rat developmental study, maternal toxicity was observed at 1,000 mg/kg/day (Thompson et. al. 1972). Fetotoxicity at all doses tested (500, 750, 1,000 mg/kg) resulted in an LEL of 500 mg/kg/day.

Developmental Effects

There is only marginally adequate testing in this area. Possible adverse developmental effects yield a LOEL of less than 500 mg/kg/day. For this review, a NOEL of 50 mg/kg/day will be used.

No developmental effects were seen in a rabbit developmental study at doses up to 400 mg/kg/day (CDFA 1986). Minor variations were observed in rats of 500 mg/kg/day exposure, the lowest dose tested. Because no NOEL seems to be determined in the available animal studies, one can be estimated by taking the lowest LEL of 500 and dividing by 10. This would result in an estimated NOEL for pilcoram developmental effects of 50 mg/kg/day, the same NOEL as in the reproduction study.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Animal studies have been interpreted as both positive and negative. Based on benign tumors in female rats, the upper 95-percent confidence limit of the cancer potency was estimated as 0.0006 per mg/kg/day.

In carcinogenicity bioassays (NCI 1978), tumors in male and female mice could not be significantly associated with doses up to 750 mg/kg/day and in male rats up to 760 mg/kg/day. However, in female rats, the incidences of benign tumors at the highest dose was suggestive of an ability of picloram to induce oncogenicity (USDA 1984).

Picloram was positive in only one traditional assay for point mutations or specific locus effects, the S. caelicolor assay for forward mutation. It was negative in all others including all strains of the

Ames assay, two Aspergillus nidulans assays, and a mouse bone marrow assay. It was positive in two assays using S. cervisae, but the meaning and utility of those assay for hazard evaluation are unclear (USDA 1984; CDFA 1986; EPA 1986). It was also negative in three assays designed to detect chromosome aberrations, a Drosophila assay, a human peripheral lymphocyte assay, and an in vivo rat bone marrow cell assay.

TRICLOPYR

Triclopyr, an oxy acetic acid compound, is a selective herbicide used for control of a variety of woody plants and broadleaved weeds.

Acute Toxicity

Based on a range of LD50's from 515 to 1,127 mg/kg, triclopyr can be considered a "moderately toxic" compound (Klaassen, 1986). In rabbit eye irritation studies, the formulation Garlon 4 is not irritating, technical triclopyr is mildly irritating, and Garlon 3A is severely irritating. In a rabbit skin irritation test, technical triclopyr is nonirritating, while Garlon 4 causes slight to moderate irritation (USDA 1984).

General Systemic Toxicity

There is marginally adequate testing in this area, but the dose at which effects are seen in animal studies varies widely. Based upon kidney effects, a NOEL of 2.5 mg/kg/day was set.

Most of the information available on triclopyr toxicity comes from unpublished work sponsored by the Dow Chemical Corporation, the manufacturer. Based on studies on mice, rats, and dogs, the liver seems to be the primary site of toxicity. In two dog studies, evidence of liver toxicity was observed at 5 mg/kg/day, and the altered kidney function was measured at 2.5 mg/kg/day. The significance of the altered kidney function is uncertain (Shipp et al. 1986).

Reproductive Effects

There is only marginally adequate testing in this area. The NOEL is greater than 30 mg/kg/day.

In a three-generation rat reproduction study, no treatment-related effects were observed up to 30 mg/kg/day, the highest dose tested. In a rat developmental study, fetotoxic effects were observed at 200 mg/kg/day, making the fetotoxic NOEL 100 mg/kg/day, the next lowest dose tested. Dam body weight gain and food consumption decreased consistently with dose (0, 50, 100, 200 mg/kg); the reduction in weight gain was not statistically significant until a dose of 200 mg/kg, with

food consumption at 100 mg/kg. The LEL for maternal effects was therefore between 50 and 100 mg/kg/day (Hanley et al. 1984; EPA 1985).

Developmental Effects

There is adequate testing in this area. Based on possible minor abnormalities involving visceral and skeletal anomalies, a NOEL of 10 mg/kg/day was set.

No adverse developmental effects were observed. In a rabbit developmental study, no maternal or developmental toxicity was observed at the highest dose tested, 25 mg/kg/day. The authors of the study considered observed incidences of visceral and skeletal anomalies to not be treatment related (Hanley et al. 1984). However, the EPA (1985) concluded otherwise, declaring that 10 mg/kg/day, the next lowest dose tested, was the appropriate NOEL for fetotoxicity.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Animal studies have been interpreted as both positive and negative for the occurrence of lung tumors. While triclopyr is considered a possible carcinogen for this analysis, no potency rating was computed.

A 2-year rat feeding study showed no oncogenic effects up to 30 mg/kg/day, the highest dose tested. According to Shipp et al. (1986), there was a significant increase in total lung tumors with increasing dose in a 2-year mouse feeding study. There seems to be some controversy about the results and interpretation of this study (EPA 1985; CDFA 1986). Triclopyr was negative in five strains of the Ames assay, negative in a bacterial DNA repair assay, and negative in a host-mediated (host was the mouse) assay for mutagenicity. It was weakly positive in one assay for chromosome effects in rats and negative in the same assay using mice (EPA 1985).

DALAPON

Dalapon, a dichlorpropionic acid, is often used in combination with atrazine for the control of grasses.

Acute Toxicity

Based on an LD50 in rats of 7,570 mg/kg, Dalapon can be considered "slightly toxic" (Klaassen, 1986). Dalapon is moderately irritating to the eyes (USDA 1984).

General Systemic Toxicity

There is marginally adequate testing in this area. Based on increased kidney weights, the NOEL is 15 mg/kg/day.

There is very little known about the systemic toxicity of dalapon. Small numbers of dogs, sheep, cattle, and hogs have been fed dalapon by capsule and oral drench. Weight loss was observed in dogs and sheep at 100 mg/kg/day. In a 2-year rat feeding study, increases in kidney weight were reported at 50 mg/kg/day. The NOEL was therefore the next lowest dose tested, 15 mg/kg/day. Microscopic examination of tissues revealed no abnormal pathology (USDA 1984).

Reproductive Effects

There is inadequate testing in this area. No NOEL is estimated.

There have been two multigeneration reproductive studies in rats and one generation reproductive study in dogs. No effects were noted in any of these studies (EPA 1984).

Developmental Effects

There is marginally adequate testing in this area. Based on decreased pup weights, a NOEL of 500 mg/kg/day was set.

The lowest NOEL from two developmental toxicity studies in rats was 500 mg/kg/day because of a reduction in pup weights at the next highest dose of 1,500 mg/kg/day (USDA 1984).

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Based on two marginally adequate studies, no evidence of oncogenic effect was seen.

Dalapon has tested for mutagenicity in four strains of the Ames assay and in an Aspergillus nidulans assay for induction of point

mutations, mitotic crossing- over and mitotic nondisjunction. All assays have been negative. A Chinese hamster ovary assay for chromosme aberration was also negative (CDFA, 1986). There have been no data reported on the potential for oncogenicity of dalapon.

ATRAZINE

Atrazine is a triazine herbicide that has had consistent use in many management programs for the selective control of grasses and forbs. Atrazine has been one of the most widely used herbicides in the United States since its introduction in 1958.

Acute Toxicity

The acute oral toxicity is 3,000 mg/kg in rats and 1,750 mg/kg in mice (USDA 1984). It thus would be considered a "moderately toxic" compound (Klaassen 1986). The LD50 for Atrex, the formulation proposed for use, is 3,080 mg/kg. Signs of acute poisoning in rats include reduced respiratory rate, motor incoordination, clonic and tonic spasms, and hypothermia (Hayes 1982). There has been one reported case by a farmer of skin allergy contracted after application of atrazine (Hayes 1982).

General Systemic Toxicity

There is marginally adequate testing in this area. Based on reduced body weights and other effects, the NOEL is 0.35 mg/kg/day.

There seem to be very few studies of the systemic toxicity of atrazine. In rats fed atrazine for 6 months, dietary levels of 100 and 500 ppm caused growth retardation, probably because of reduction in food intake. A 2-year dog feeding study resulted in decreases in body weight at 1,500 ppm, resulting in a NOEL of 150 ppm (3.75 mg/kg/day), the next lowest dose tested. A recently completed 2-year feeding study with rats resulted in a systemic NOEL of 7 ppm (0.35 mg/kg/day) based on reduced body weight, reduced clinical blood parameters, and decreased glucose levels at the next highest dose (CDFA 1986).

Reproductive Effects

There is only marginally adequate testing in this area. The NOEL is greater than 5 mg/kg/day.

No multigeneration reproductive studies on unformulated atrazine are available. There has been one three-generation rat study using Atrazine 80W. No effects were observed at the highest dose tested (5 mg/kg) (EPA 1985; CDFA 1986). There have been several experiments evaluating the effect on reproduction of atrazine administered during

gestation. In one, the dose was oral, up to 100 mg/kg/day, and no effects were observed. In the second, dosage was made by subcutaneous injection on days 3, 6, and 9 of gestation. Dosage as high as 200 mg/kg/day by this route did not affect the number of pups per litter nor their weight at weaning (Peters and Cook 1973). In rats administered atrazine by gavage, fetotoxicity was observed at 10 mg/kg/day, the lowest dose tested.

Developmental Effects

There is marginally adequate testing in this area. In a rabbit developmental study, fetotoxicity was observed at 75 mg/kg, leaving the fetotoxic NOEL at 5 mg/kg/day, the next lowest dose tested (CDFA 1986).

Neurotoxic Effects

There is only marginally adequate data for a qualitative assessment. There is weak to moderate evidence of some neurotoxic effect in animal experiments.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Based on mammary tumor formation in rats, the upper 95-percent confidence limit of the cancer potency was estimated as 0.2 per mg/kg/day.

Atrazine was not oncogenic in an 18-month study of mice dosed at 21.5 mg/kg/day (EPA 1985). However, there was a significant increase in total mammary tumors for Sprague-Dawley rats fed 70, 500, and 1,000 ppm atrazine for 2 years (CDFA 1986). Two positive tests for cell transformation (EPA 1985) provided further evidence that atrazine may be oncogenic.

Atrazine has been tested in 38 short-term assays for genotoxicity. The picture that emerges is one of a chemical that by itself is not strongly genotoxic. It was only positive in 4 of 18 assays for point mutations (negative in five strains of the Ames assay with and without animal-derived activation material). It was positive in 7 of 20 assays measuring different aspects of potential chromosome damage (CDFA 1986; NAS 1977).

However, of the 11 positive assays, 2 of the 4 assays for point mutations and 2 of the 7 assays for chromosome damage were done either in systems using plant-derived activation material or plant-derived models for genotoxicity. In addition, a DNA damage assay

using human cells was positive with a plant-derived activation material. The data suggest that plant material can metabolize atrazine into a mutagen. This raises concern about the presence of atrazine in the environment, such as in water supplies (NAS 1977).

2,4-DP

2,4-DP is a chlorophenoxy compound that has been used, generally in combination with 2,4-D, for control of shrubs and broadleaved weeds.

Acute Toxicity

Based on the LD50 for rats in the range of 446 to 633 mg/kg (EPA 1984), 2,4-DP can be considered a "moderately toxic" to "very toxic" compound (Klaassen 1986). Signs of poisoning in rats include hypersensitivity and decreased motor activity.

General Systemic Toxicity

There is adequate testing in this area. Based on a wide range of effects, the NOEL is set at 5 mg/kg/day.

Chronic and subchronic feeding studies with rats and mice have shown 2,4-DP to affect blood chemistry, liver, and other organ weights and general body weight at doses from 25 to 300 mg/kg/day. The lowest NOEL reported so far is 5 mg/kg/day in a subchronic rat feeding study (EPA 1984).

Reproductive Effects

There is only marginally adequate testing in this area. Based on fetotoxicity, the NOEL is set at 6.25 mg/kg/day.

Fetotoxicity was observed in a three-generation rat study at 25 mg/kg, leaving the NOEL at 6.25 mg/kg/day, the lowest dose tested (EPA 1984).

Developmental Effects

There is adequate testing in this area. Based on minor to major abnormalities, a LOEL of 25 mg/kg/day was observed. No NOEL was observed, and a NOEL of 2.5 (1/10 the LOEL) was used in this analysis.

There seems to be evidence that 2,4-DP is a developmental toxicant. While no maternal or fetotoxic effects were observed at 25 mg/kg/day in rabbits fed 2,4- DP during gestation, displaced kidneys, navel hernia, and distorted ribs were observed in the pups at that dose. No lower doses were included.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Based on mixed tumors (primarily in the thyroid and pituitary glands) among male rats, the upper 95-percent confidence limit of the cancer potency was estimated as 0.06 per mg/kg/day.

For epidemiology results, see the discussion under 2,4-D.

2,4-DP has induced tumor formation in one of three animal studies. No tumors were found in mice at doses of up to 300 mg/kg per day or in Fischer F-334 rats at doses up to 150 mg/kg/day. However, in a study with Sprague-Dawley rats, tumors of either the pituitary, thyroid, or brain were found at all doses tested (25 mg/kg to 200 mg/kg) (EPA 1984). 2,4-DP only has been tested in bacterial mutagenicity assays with mixed results. A three-strain Ames assay was negative, both with and without S-9.

HEXAZINONE

Hexazinone, a triazine herbicide, is used to control shrubs, grasses, and forbs.

Acute Toxicity

Based on a rat LD50 of 1,690 mg/kg, hexazinone can be classified as "moderately toxic" (Klaassen 1986). Signs of poisoning in rats include tremors, irregular respiration, lethargy, convulsions, congestion, and prostration (USDA 1984). Hexazinone has tested negative for skin sensitization but positive for eye irritation (USDA 1984).

General Systemic Toxicity

There is only marginally adequate testing in this area. Based on liver effects, the NOEL is set at 10 mg/kg/day.

Subchronic and chronic studies in dogs, rats, and mice have shown some liver effects (increased weight, hyperplasia, and focal necrosis), usually at high doses, but no other toxicity has been attributed to hexazinone. The lowest NOEL from any systemic study is 200 ppm (10 mg/kg/day) (EPA 1984; USDA 1984).

Reproductive Effects

There is only marginally adequate testing in this area. Based on reduced pup weights, the NOEL is set at 50 mg/kg/day.

A three-generation reproduction study in rats was negative, except for decreased fetal body weight in the highest dose tested (125 mg/kg/day).

Developmental Effects

There is only marginally adequate testing in this area. Based on minor abnormalities (skeletal variants), a NOEL of 50 mg/kg/day was set.

Rat and rabbit developmental studies have been negative up to 50 mg/kg (Kennedy and Kaplan 1984).

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is adequate testing in this area. Based on two adequate studies, no oncogenic effect was observed.

A mouse chronic study and a rat chronic study both were negative for oncogenicity. Hexazinone was negative in a five-strain Ames assay, an in vitro mammalian cell assay, an assay of unscheduled DNA repair synthesis in mammalian somatic cells, and a mammalian cytogenicity assay. It was positive in an in vitro cytogenicity assay with rodent cells at high doses (USDA 1984).

FOSAMINE

Fosamine, a carbamate compound, is not currently registered for forestry applications.

Acute Toxicity

Fosamine is a mild to moderate skin irritant but does not seem to be a dermal sensitizer (EPA 1985). Based on an LD50 of 24,400 mg/kg for Krenite, the formulated product proposed for use, Krenite seems to be a "practically nontoxic" compound (Klaassen 1986). Signs of poisoning in rats include respiratory distress and diarrhea (USDA 1984).

General Systemic Toxicity

There is marginally adequate testing in this area, but the dose at which effects are seen in animal studies varies widely. Based on increased stomach weight, the NOEL is 25 mg/kg/day.

Subchronic studies with dogs, rats, and sheep recorded only diarrhea or increased stomach weights up to 10,000 ppm. The NOEL for the study was 25 mg/kg/day because of increased stomach weight at 250 mg/kg/day, the next highest dose tested.

Reproductive Effects

There is inadequate testing in this area. No NOEL was estimated.

Developmental Effects

There is inadequate testing in this area. No NOEL was estimated. Teratologic effects (minor malformations–hydronephrosis) were detected in rats at 10,000 ppm (207 mg/kg/day).

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is inadequate testing in this area. No potency level was estimated.

Chromosome damage was observed in a Chinese hamster ovary cell in an in vitro cytogenetic assay, but fosamine was negative in an Ames assay, an in vivo bone marrow cytogenetic assay in rats, a chinese hamster ovary study, and an assay for unscheduled DNA synthesis. No chronic studies have been carried out with fosamine formulations or ammonium fosamine (USDA 1984).

DICAMBA

Dicamba, an arylaliphatic acid, is a relatively nonselective herbicide used against a variety of broadleaved weeds and brush species.

Acute Toxicity

The acute toxicity of dicamba in rats ranges from 757 to 2,900 mg/kg, which indicates that dicamba is a "moderately toxic" compound (Klaassen 1986). Signs of acute poisoning in animals include muscle spasms, bradycardia, and inhibited voluntary and involuntary reflexes

(NAS 1977). Exposed workers have reported muscle cramps, dypsnea, nausea, vomiting, skin rashes, and loss of voice or swelling of cervical glands (EPA 1987).

General Systemic Toxicity

There is generally inadequate testing in this area. Subchronic and chronic studies on rats, dogs, and mice have revealed only slight liver necrosis or liver weight changes. Based on decreased body weight, a NOEL of 0.125 was set.

According to the EPA (1985), the lowest NOEL of 25 mg/kg is from a rat subchronic feeding study. The National Academy of Sciences reports NOELS of 10.3 mg/kg and 19.3 mg/kg from rat feeding studies. Interestingly, effects seen at lower doses in subchronic studies with rats have not been observed in chronic studies at doses up to 125 mg/kg (NAS 1977). A 2-year dog feeding study, completed in 1960, reported effects at doses as low as 0.125 mg/kg/day. However, there are aspects of this study that cast doubt on its quality and usefulness in evaluating risk (EPA 1986).

Reproductive Effects

There is only marginally adequate testing in this area. A NOEL of greater than 500 mg/kg/day was set.

No reproductive effects were seen in two three-generation rat reproductive studies.

Developmental Effects

There is marginally adequate testing in this area. Based upon observed skeletal malformations, a NOEL of 3 mg/kg/day was set.

No developmental effects have been observed in a number of teratology studies. A pilot teratology study in rabbits showed potential maternal and fetotoxic effects at doses as low as 1.0 mg/kg/day. The complete rabbit teratology study that followed showed maternal and some fetotoxic effects at 10 mg/kg, leaving the lowest NOEL for developmental effects at 3.0 mg/kg/day, the next lowest dose tested (CDFA 1986).

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is marginally adequate testing in this area. Three chronic studies, two in rats and one in dogs, were negative for oncogenicity (EPA 1985, 1986). Dicamba tested negatively in a five-strain Ames assay and three other bacterial mutagenicity assays. It was weakly positive in one unscheduled DNA synthesis assay and negative in a second one (CDFA 1986). Based upon thyroid carcinomas and malignant lymphomas in male rats, dicamba may be a carcinogen. No cancer potency level has been estimated.

ASULAM

Asulam is a carbamate compound used as a selective postemergence systemic herbicide.

Acute Toxicity

Unlike some other carbamate herbicides (Murphy 1986), asulam does not seem to be a skin-sensitizing agent in humans. Based on an LD50 greater than 5,000 mg/kg in rats, asulam can be considered as slightly toxic (Klaassen 1986).

General Systemic Toxicity

There is marginally adequate testing in this area. Based on effects on the liver and changes in organ weights, a NOEL of 50 mg/kg/day has been estimated.

Chronic and subchronic studies on dogs, rats, and mice have shown very little systemic toxicity beyond increases and decreases in organs weights. The lowest NOEL of any study was 50 mg/kg, determined in a 107-week rat feeding study (EPA 1985).

Reproductive Effects

There is only marginally adequate testing in this area. A NOEL of 50 mg/kg/day was set.

In one multigeneration reproductive study, fetotoxic effects were observed at 5,000 ppm, leaving the NOEL at the next lowest dose of 1,000 ppm (50 mg/kg) (EPA 1985).

Developmental Effects

There is only marginally adequate testing in this area. Based on minor abnormalities, a NOEL of 300 mg/kg/day was set.

Two developmental toxicity studies (rat and rabbit) revealed no developmental effects from dietary feeding of asulam.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is adequate testing in this area. Based on thyroid tumors in male rats, the upper 95-percent confidence limit of the cancer potency was estimated as 0.02 per mg/kg/day.

Asulam seems to be a thyroid carcinogen in rats but not to be carcinogenic in mice. Two mice studies with doses up to 5,000 ppm showed no oncogenicity, while a significant increase in adrenal medullary hyperplasia, thyroid cell carcinomas, and parafollicular cell carcinomas was observed in female rats only at doses of 50, 250, and 1,250 mg/kg/day. Based on three short-term tests, asulam does not seem to be a direct-acting (that is, mutagenic) carcinogen. It was negative in a five-strain Ames assay, a cell transformation assay (C3H/10T 1/2 CL8 cells) and a dominant lethal assay (mice) (EPA 1985; CDFA 1986).

TEBUTHIURON

Tebuthiuron, a substituted urea, is used for the control of woody vegetation and annual weeds.

Acute Toxicity

Based on an acute oral LD50 in rats between 296 and 720 mg/kg (EPA 1986), tebuthiuron can be considered "moderately to very toxic" (Klaassen 1986). Symptoms of poisoning in the rat included ataxia, vomiting, tremors, and convulsions (EPA 1986). Tebuthiuron seems to be an eye irritant, based on corneal opacity and eye irritation in rabbits exposed to a 60-percent solution (EPA 1986).

General Systemic Toxicity

There is marginally adequate testing in this area, but the dose at which effects are seen in animal studies varies widely. While general toxic effects involve weight suppression, the NOEL of 12.5 mg/kg/day is based on increased thyroid and spleen weights.

Chronic and subchronic studies demonstrate that large doses (100 ppm to 1,000 ppm) of tebuthiuron cause growth suppression and body weight suppression in rats, cattle, and hens. The lowest NOEL of the available systemic studies is 12.5 mg/kg for a 90-day study in which

dogs experienced increased thyroid and spleen weights at 25 mg/kg (EPA 1986).

Reproductive Effects

There is only marginally adequate testing in this area. A NOEL of less than 20 mg/kg/day was set.

One two-generation reproduction study in rats reported a NOEL of 100 ppm (5 mg/kg/day) (Hoyt et al. 1981), the highest dose tested, while a three-generation reproduction study in rats reported a reproductive NOEL of less than 400 ppm (20 mg/kg/day), the lowest dose tested (EPA 1986).

Developmental Effects

There is only marginally-adequate to inadequate testing in this area, and the dose at which effects are seen in animal studies varies widely. Based on possible minor abnormalities, a NOEL of greater than 90 mg/kg/day has been estimated.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Two marginally adequate studies demonstrated no oncogenic effect.

A chronic rat and a chronic mouse study were negative for oncogenicity up to 1,000 ppm, the highest dose tested (EPA 1986). There is little information available to judge the mutagenic potential of tebuthiuron. A five-strain Ames assay was negative. A test for induction of forward mutation in mouse lymphoma cell with metabolic activation was negative, but the same assay without metabolic activation was weakly positive.

DIURON

Acute Toxicity

Diuron is a compound considered to be "moderately" toxic based on a rat LD50 of 3,400 mg/kg (Klaassen 1986; Hayes 1982). Signs of poisoning in the rat include ataxia, drowsiness, hypothermia, glycosuria, proteinuria, aciduria, and respiratory failure (Hayes 1982). Ten daily doses to rats of 1,000 mg/kg depressed weight and increased production of red blood cells but were not fatal. Diuron may

irritate the skin, eye, and nose. A 39-year-old woman ingesting a diuron-amitrole mixture of 38 mg/kg diuron showed no signs of intoxication (Hayes 1982).

General Systemic Toxicity

There is generally inadequate testing in this area. However, effects have been observed including reduced organ weights and blood abnormalities. Based on blood abnormalities, a NOEL of 0.625 mg/kg/day was observed.

A 2-year dog study and a 2-year rat study both had systemic NOEL's of 25 ppm (0.625 mg/kg/day). The toxic effect at the next highest dose was a trace of an abnormal blood pigment, which was a sulfhemoglobin. At higher doses, hyperplasia of bone marrow, increased blood products, and abnormal spleenic weight were observed (Hodge et al. 1967).

Reproductive Effects

There is inadequate testing in this area. No NOEL was estimated.

Developmental Effects

There is only marginally adequate testing in this area. Based on minor abnormalities (delayed ossification) at the lowest tested dose, yielding a LOEL of 125 mg/kg/day, a NOEL of 12.5 mg/kg/day (1/10 the LOEL) has been estimated.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is inadequate testing in this area. No cancer potency level has been calculated.

No oncogenic effects were reported in two chronic studies (Hodge et al. 1967; EPA 1986). Diuron was negative in an Ames assay, a CHO/HGPRT forward gene mutation assay, and an unscheduled DNA synthesis in primary rat hepatocytes assay. It was positive in an in vivo cytogenicity assay, suggesting that it is clastogenic (EPA 1986).

SIMAZINE

Simazine, a triazine compound, is used for selective control of annual and perennial grasses and broadleaved weeds.

Acute Toxicity

The oral LD50 in rats of simazine technical material is greater than 5,000 mg/kg; the LD50 of Princep, one of the formulations proposed for use, is greater than 15,380 mg/kg. Thus Simazine seems to be a "practically nontoxic" compound (Klaassen 1986).

General Systemic Toxicity

There is only marginally adequate testing in this area. A variety of effects are seen. Based on blood cell counts, a NOEL of 5 mg/kg/day was estimated

However, some mortality has been reported at 50 mg/kg/day in a 28-day rat feeding study using crystalline material and at 10 mg/kg in a 28-day rat feeding study using technical material. In both studies, at 2,500 mg/kg, hyperplasia, ulcers, and fissures of the stomach were observed. In a third 21-day rat feeding study, blood parameters and kidney effects were observed at 10 mg/kg/day, the lowest dose tested (EPA 1986).

Reproductive Effects

There is inadequate to marginally adequate testing in this area, and the estimated NOEL is not considered stable. A NOEL of greater than 5 mg/kg/day was estimated.

A three-generation reproductive study in rats showed no effects at 100 ppm of simazine 80W (5 mg/kg), the only dose tested (EPA 1986).

Developmental Effects

There is adequate testing in this area. A NOEL of 5 mg/kg/day was estimated.

In a developmental study using rabbits, maternal toxicity was observed at 75 mg/kg/day. Fetotoxicity was observed at 75 and 200 mg/kg, the highest dose tested.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is inadequate testing in this area. No cancer potency level has been calculated.

No effects were seen in a 2-year rat feeding study using a 50-percent simazine solution at 5 mg/kg/day, the highest dose tested. In a chronic mouse feeding study, no tumors were observed at a dose of 215 mg/kg (Innes 1969), the only dose tested. This information is not sufficient to evaluate the carcinogenic potential of simazine. Simazine was negative for mutagenicity in four strains of the Ames assay, two S. cervisae assays, two mammalian cell culture assays, and a host-mediated assay using mice. It also was negative in three bacterial and two mammalian cell DNA damage/repair assays. It was positive in three Drosophila assays for chromosomal aberrations but negative in a Chinese hamster micronucleus test. It also was positive for sister chromatid exchange in human lymphocytes and in a plant assay (CDFA 1986). In summary, there seems to be limited evidence for the potential of simazine to cause chromosomal damage.

BROMACIL

Bromacil is a substituted uracil, which has had limited use for the control of a broad spectrum of broadleaved weeds, grasses, and woody shrubs.

Acute Toxicity

The acute LD50 of bromacil in rats is 5,200 mg/kg (NAS 1977). This indicates that bromacil is a "slightly toxic" compound (Klaassen 1986). The signs of poisoning in rats include rapid respiration, prostration, discomfort, and initial weight loss.

General Systemic Toxicity

There is only marginally adequate testing in this area. A variety of effects are seen. Based on weight loss and liver and thyroid effects, a NOEL of 6.25 mg/kg/day was set.

There are a small number of animal studies available on bromacil. Subchronic studies in rats revealed hyperplasia of the liver and increased thyroid activity at doses of 250 mg/kg/day and greater. A 2-year rat feeding regime resulted in weight retardation and slight hyperplasia in the thyroid at 1,250 ppm, leaving the NOEL at 250 ppm, the next lowest dose tested (Sherman and Kaplan 1975). A 2-year dog study had similar results, with a NOEL of 250 ppm or 6.25 mg/kg/day (Sherman and Kaplan 1985; NAS 1977; EPA 1985).

Reproductive Effects

There is only marginally adequate testing in this area. A NOEL of greater than 12.5 mg/kg/day was estimated.

One reproductive rat feeding study showed no effects at one dose tested, 12.5 mg/kg/day (Sherman and Kaplan 1975).

Developmental Effects

There is marginally adequate testing in this area. A NOEL of 12.5 mg/kg/day was estimated.

Two developmental studies, a rabbit feeding study (Sherman and Kaplan 1975) and a rat inhalation study (EPA 1985), showed no effects.

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is only marginally adequate testing in this area. Based on liver tumor formation in male mice, the upper 95-percent confidence limit of the cancer potency was estimated as 0.004 per mg/kg/day.

In a 2-year feeding study with CD-1 mice, liver tumors were observed at a dose of 5,000 ppm (750 mg/kg/day). The previously described 2-year study with rats was negative for oncogenicity (Sherman and Kaplan 1975; NAS 1977; EPA 1985). There have been over a dozen short-term assays for genotoxicity using bromacil. Bromacil was positive for a mouse lymphoma L5178Y forward mutation and for a Drosophila sex-linked recessive lethal test. It was negative in the Ames and Escherichia coli mutation assay and in the Sacchromyces mutation test, for E. coli pol A and Bacillus subtilis DNA and Salmonella typhimurium differential growth, for synthesis in WI-38 cells, for sister chromatid exchange in Chinese hamster cells, for a mouse micronucleus test, and for a mouse dominant lethal test. Thus, there is some evidence that bromacil may be a mutagen in eukaryotic systems but no evidence for chromosomal damage (CDFA 1986).

AMITROLE

Amitrole is an aminotriazole used as a nonselective herbicide. Used at one time for cultivation of such food crops as cranberries (Hayes 1982), as of 1973, it has not been permitted for use on crops or pasture land where food for humans or animals may be contaminated (Gaines et al. 1973; Murphy 1986).

Acute Toxicity

Amitrole can be described as moderately to slightly toxic (Klaassen 1986), depending on formulation. The LD50 of the active ingredient

fed to rats ranges from 1,100 to 25,000 mg/kg. Signs of poisoning include intestinal paralysis, pulmonary edema, and hemorrhages of various organs (Hayes 1982).

Because many plant species can metabolize amitrole to a nontoxic form, ammonium thiocyanate is often added to formulations. Ammonium thiocyanate serves to block toxicity-reducing metabolism in plants. Because the LD50 of ammonium thiocyanate is around 700 mg/kg in the rat, toxicity of formulations containing ammonium thiocyanate, such as Amitrol-T, can be increased. Other potential acute hazards with amitole include eye injury reported for amitrole and the formulation Amizol by human workers. Rabbits treated with 0.2-percent Amizol have contracted cataracts, confirming the potential for eye injury hazard from amitrole exposure. There has been one human exposure reported. A 39-year-old woman drank an amitrole-diuron mixture containing 20 mg/kg amitrole with no apparent ill effects (USDA 1984).

General Systemic Toxicity

There is only marginally adequate testing in this area. A variety of effects are seen. Based on thyroid effects, a NOEL of 0.025 mg/kg/day was set.

Both subchronic and chronic studies with rats and mice show that amitrole affects the thyroid (Hayes 1982) at low doses. The lowest NOEL reported from over a dozen subchronic and chronic animal toxicity studies is 0.025 mg/kg/day (EPA 1984).

Reproductive Effects

There is inadequate testing in this area, but effects were seen and an unreliable NOEL of 5 mg/kg/day was estimated.

Effects on reproduction in rats have been studied by Gaines et al. (1973). In a two-generation study in rats, at dietary levels of 1,000 ppm and 500 ppm, the pups that were born were smaller and had atropic thymuses and spleens indicative of runt disease. Most of them died within a week of weaning. Reproduction was not affected at dietary levels of 100 ppm and 25 ppm.

Developmental Effects

There is adequate testing in this area. Based on possible minor abnormalities (structural changes), a NOEL of 4 mg/kg/day was estimated.

The EPA (1984) lists two mouse developmental toxicity studies that showed no teratogenic effects at the highest doses tested. While

studies on rats have shown no adverse developmental effects (CDFA 1986), there are data to suggest that the thyroids of fetuses may be affected by doses of as low as 0.4 mg/kg/day to the dams (Shalette et al. 1963). A recent developmental study on rabbits indicated increased abortions and decreased weight gain in the dams and increased structural changes in the offspring at a dose of 40 mg/kg/day, leaving the NOEL at the next lowest dose of 4 mg/kg/day (CDFA 1986). Based on these animal models, there is a possibility that a developing fetus may experience some thyroid effects from a high dose to the mother (Gaines et al. 1973).

Neurotoxic Effects

There is inadequate data for a qualitative assessment.

Immunotoxic Effects

There is inadequate data for a qualitative assessment.

Oncogenicity

There is marginally adequate testing in this area. Based on thyroid tumors in rats, the upper 95-percent confidence limit of the cancer potency was estimated as 1.4 per mg/kg/day.

Three epidemiology studies have been published linking amitrole to human cancer deaths. The international cancer research group, the International Agency for Research on Cancer (IARC), stated in 1982 that the evidence is insufficient to establish an association between amitrole and human cancers (IARC 1982). Three epidemiology studies have been published linking amitrole to death in humans because of cancer, but the IARC describes this evidence as "inadequate." (IARC 1974, 1982) Based on two mice studies and three rats studies showing amitrole to produce thyroid and liver tumors following oral and subcutaneous administration, IARC (1982) has described the evidence for carcinogenicity to animals as "sufficient." Amitrole seems to be an inducer of cell transformation; however, it is inactive as a mutagen in bacterial systems. While there is some evidence that it may act via a mutational mechanism, most investigators do not consider amitrole to be genotoxic (IARC 1982; Barrett 1987).

Characterization and
Management of Risk

Glossary

Glossary

A

Absorption—The taking up of liquids by solids or the passage of a substance into the tissues of an organism as the result of several processes; that is, diffusion, filtration, or osmosis.

Active ingredient (a.i.)—The effective chemical component of a pesticide formulation.

Acute toxicity—The potential of a substance to cause injury or illness when given in a single dose or in multiple doses over a period of 24 hours or less.

Adsorption—Adhesion of substances to the surfaces of solids or liquids. Technically, the attraction of ions of compounds to the surfaces of solids or liquids.

Anemia—deficiency in red blood cells.

Angiosarcoma—A malignant tumor of the blood vessel.

Anorexia—Prolonged loss of appetite.

Assay—A test or measurement used to evaluate a characteristic of a chemical. See bioassay, mutagenicity assay.

B

BMP (best management practice)—A practice, or combination of practices, that is determined by a State (or designated area-wide planning agency) after problem assessment, examination of alternative practices, and appropriate public participation to be the most effective practicable means (including technological, economic, and institutional considerations) of preventing or reducing the amount of pollution generated by nonpoint sources to a level compatible with water quality goals.

Bioaccumulation—The process of a plant or animal selectively taking in or storing a persistent substance. Over a period of time, a higher concentration of the substance is found in the organism than in the organism's environment.

Bioassay—A method for quantitatively determining the concentration of a substance by its effect on the growth of a suitable animal, plant, or microorganism under controlled conditions.

Biomass—The total of living organisms of one or more species per unit area or of all the species in a community measured in dry weight or kilocalories.

Boom (herbicide spray)—A tubular metal device that conducts an herbicide mixture from a tank to a series of spray nozzles. A boom may be mounted beneath a helicopter or a fixed-wing aircraft or behind a tractor.

Broadcast burning—Allowing a controlled fire to burn over a designated area within well-defined boundaries.

C

Cancer potency—A measure of the relative ability of a substance to cause cancer. Usually expressed as a function of unit dose as per mg/kg/day. When multiplied by the estimated daily lifetime dose (in mg/kg/

day) of an individual, it will yield an estimate of the probability of that individual developing cancer.

Carcinogen—Any cancer-producing substance.

Chemical degradation—The breakdown of a chemical substance into simpler components through chemical reactions.

Chronic toxicity—The adverse effects of a series of small doses of a substance over a long period that may be related to changes in appetite, growth, metabolism, reproduction, and life span.

Conjunctivitis—Inflammation of the mucous membrane that lines the inner surface of the eyelids.

DNA—Deoxyribonucleic acid. The nucleic acid containing the sugar deoxyribose, which is the molecular basis of heredity in most organisms.

Degrade—To decompose or break up.

Demyelination/demyelinization—The destruction or removal of the myelin sheath of nerve tissue. The myelin sheath is composed of layers of myelin, a lipid material that provides electrical insulation and protection for the neuron.

Dermal exposure—The portion of a toxic substance that an organism receives as a result of the substance coming into contact with the organism's body surface.

Dermatitis—Inflammation of the skin.

Developmental Toxicity--The adverse effects on the developing organism that may result from exposure prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may include: 1) lethality in the developing organisms, 2) structural abnormalities, 3) altered growth, and 4) functional deficiency (USEPA 1986).

Dominant lethal test—A test to detect a mutation of a dominant gene that may be fatal to the next generation.

Dose—A measurable quantity of an herbicide or other substance.

Drift—That portion of a sprayed chemical that moves off a target site because of wind.

Edema—An excessive accumulation of fluid in the cells, tissue spaces, or body cavities resulting from a disturbance in the fluid exchange mechanism.

Embryotoxic—The adverse effects occurring at the early stages of development (embryonic period).

Environmental fate—The transport, accumulation, and disappearance of an herbicide in the environment.

Epidemiology—A science that deals with the incidence, distribution, and control of disease in a population.

Exposure analysis—The estimation of the amount of chemicals that organisms receive during the application of pesticides.

Fetotoxic—The adverse effects occurring at the later stages of development (fetal period).

Fibroadenoma—an abnormal growth of fribrous tissue.

Forest Service manual (FSM)—An internal set of operating directives that governs Forest Service activities.

Formulation—The form in which a pesticide is packaged or prepared for use. A chemical mixture that includes a certain percentage of active ingredient (technical chemical) often with an inert carrier.

Fuel—Any substance or composite mixture that can ignite and burn.

Girdling—Making continuous incisions around a living stem through at least both bark and cambium, generally resulting in the death of the tree.

Half-life—The time required for a substance (such as an insecticide) in or introduced into a living or nonliving system to be reduced to half of its original amount, whether by excretion, metabolic decomposition, or other natural process.

Hazard analysis—The determination of whether a particular chemical is or is not causally linked to particular harmful effects.

Herbicide—A chemical used to control, suppress, or kill plants, or to severely interrupt their normal growth processes.

Heritable—Capable of being inherited or of passing to others by inheritance.

Hydrocephaly—abnormal fluid surrounding the brain.

Hypoactivity—reduction in normal activity.

LD_{50}—Median lethal dose. The size of a single dose of a chemical necessary to kill 50 percent of the organisms in a specific test situation. It is usually expressed in milligrams of the chemical per kilogram of body weight (mg/kg). It may be fed (oral LD_{50}), applied to the skin (dermal LD_{50}), or administered in the form of vapors (inhalation LD_{50}).

LEL—Lowest effect level. Frequently used when an effect is observed at the lowest level tested, so there is no NOEL. It differs from LOAEL in that the effect need not be considered adverse.

LOAEL—Lowest observed adverse effect level. Lowest dose level at which effects are observed.

LOEL—Lowest observed effect level. Same as LEL.

mg/kg—Milligrams per kilogram. Used to designate the amount of toxicant required per kilogram of body weight of test organisms to produce a designated effect; usually the amount necessary to kill 50 percent of the test animals. 1 mg/kg = 1 ppm. 1 mg = 0.000035 ounce. 1 kg = 2.2 pounds.

mg/kg/day—Milligrams per kilogram of body weight per day.

Metabolite—A product of the chemical changes in living cells that provides energy and assimilates new material.

Microbial degradation—The breakdown of a chemical substance into simpler components by bacteria.

Microgram—1 millionth of a gram. Abbreviated as ug.

Mitigate—To make less harsh or harmful.

Mutagen—A substance that tends to increase the frequency or extent of genetic mutations (changes in hereditary material).

Mutagenicity assay—a study to determine if a substance causes genetic damage.

Mutation—a change in the genetic material of a cell.

Myotonia—muscle spasm.

NOEL—The no-observed-effect level. In a series of dose levels tested, it is the highest level at which no effect is observed; that is, safe in the species tested.

Neuropathy—Any disease affecting neurons, the fundamental functional unit of nervous tissues.

Neurotoxic—Toxic to nerves or nervous tissue.

Oncogenic—Capable of producing or inducing tumors, either benign (noncancerous) or malignant (cancerous), in animals.

Ossification—the formation of bone.

ppb (parts per billion)—The number of parts of a substance per billion parts of a given material. 1 ppb = 1 ug/liter (water or air).

ppm (parts per million)—A unit for measuring the concentration of a substance (such as a pesticide) in a carrier medium (such as food or water). For example, where the concentration is 1 ppm, the weight of the substance is 1 millionth the weight of the carrier medium; thus, 1 ppm is equal to 1 milligram of substance per kilogram of food or organism body weight, and it is equal to 1 milligram of substance per liter of water.

Particulates—Finely divided solid or liquid particles in the air or in an emission; includes dust, smoke fumes, mist, spray, and fog.

Pesticide—Any substance or mixture of substances used in controlling insects, rodents, fungi, weeds, or other forms of plant or animal life that are considered to be pests.

Pharmacokinetics—The study of rates of absorption, metabolic breakdown, and excretion of chemicals in animals.

Pheochromocytoma—tumor of the adrenal gland.

Phytotoxic—Poisonous or harmful to plants.

Polyneuritis—multiple degenerative lesions of the nerve.

Prescribed burning—The use of fire as a management tool under specified conditions for burning a predetermined area.

Recessive lethal test—A test to detect a mutation of a recessive gene that may be fatal to the next generation.

Release—Freeing a tree or group of trees from competition by cutting or otherwise eliminating growth that is overtopping or closely surrounding it.

Residue level—Amount of pesticide that may remain on a crop after harvesting.

Resorption—absorption of fetal implant into the uterine wall.

Risk analysis—The description of the nature and often the magnitude of risk to organisms, including attendant uncertainty.

Safety factor—A factor conventionally used to extrapolate human tolerances for chemical agents from no-observed-effect levels in animal test data.

Sister chromatid exchange assay—Mutation assay designed to evaluate an alteration in the normal exchange of genetic material.

Site preparation—The removal of slash and/or competing vegetation and usually the exposure of bare mineral soil to prepare an area for regeneration.

Slash—The residue left on the ground after timber cutting and/or accumulating as a result of storm, fire, or other damage. It includes unused logs, uprooted stumps, broken stems, branches, twigs, leaves, bark, and chips.

Stand—An aggregation of trees or other growth occupying a specific area and sufficiently uniform in species composition, age, arrangement, and other conditions to be distinguishable from the forest, other growth, or other land cover on adjoining areas.

Standard deviation—The positive square root of the variance around a mean (average). With normally distributed data about 96 percent of the observation will lie within 2 standard deviations of the mean.

Teratogen—A substance tending to cause developmental malformations in unborn human or animal offspring. Teratogenicity is the capacity of a substance to cause anatomical, physiological, or behavioral defects in animals exposed during embryonic development.

Threshold—A dose or exposure below which there is no apparent or measurable adverse effect.

Threshold limit value (TLV)—The concentration of an airborne constituent to which workers may be exposed repeatedly, day by day, without adverse effect.

Toxic—Poisonous.

Volatility—The tendency of a substance to evaporate at normal temperatures and pressures.

Volatilization—The vaporizing or evaporating of a chemical substance.

Potential Toxic Effects Summary

Key to Potential Toxic Effects Summary

	Acute Toxicity: Oral LD50 (Mg/Kg of Body weight)	Mutagenicity Carcinogenicity	Systemic Observed Damage to Vital Functions	Reproductive/ Developmental
High	25 or Less	Mutagenic, confined carcinogenic [EPA Class A and B]	Irreversible	Adverse effects in rodents & non-rodents or conclusive in one species
Moderate	25-250	Weakly mutagenic some evidence of carcinogenicity [EPA Class C]	Serious and reversible	Suspected adverse effects in one species
Low	250-1000	Weakly mutagenic negative cancer test	Transient and not permanent	No adverse effects
Negligible	Greater than 1000	Negative mutagenic and cancer test results [EPA Class D]	None	

Insufficient Information

Key for Probable Effects to the Public, Probability of Worker Dose Effect, Probability of Worker Reproductive Effect

Probability of exposure to a toxic concentration	Calculated margin of safety
High	Less than 10
Moderate	Between 10 and 100
Low	Between 100 and 1000
Negligible	Greater than 1000

Margin of Safety is the number of times the NOEL for animals exceeds the expected dose.
NOEL is no observable effects level.

Quality of Information

I = Inadequate information available for evaluating toxicity
M–I = Marginal but usable information available for evaluating toxicity—widely varying results provide an unstable assessment
M = Marginal but usable information available for evaluating toxicity—additional studies may significantly change assessment
A = Adequate information available—more studies unlikely to change assessment

	Program Use	Summary of Potential Health Hazards				Probable Effects on the Public				Risk Concern	Probability of Workers Receiving a Dose Which Affects General Health				Probability of Workers Receiving a Dose Which Affects Reproduction				Quality of Information						Health Risk	Risk Management
						To General Health		On Reproduction																		
		Poisoning	Systemic	Cancer	Reproductive	Routine	Large Spill	Routine	Large Spill		Aerial Mixer/Loader	Backpack Sprayer	Right-of-Way Mixer/Loader	Hack-and-Squirt	Aerial Mixer/Loader	Backpack Sprayer	Right-of-Way Mixer/Loader	Hack-and-Squirt	Systemic	Cancer	Reproductive	Developmental	Neurological	Immunological		
2,4-D	Broadleaved weeds and herbaceous plants	LOW	HIGH	LOW MOD INS	HIGH	MOD	HIGH	MOD	HIGH	MOS =< 100	MOD	HIGH	MOD	MOD	LOW	MOD	LOW	MOD	A	M	A	M	A	M	Moderate probability of workers receiving a dose that may cause reproductive or developmental effects in operations. Moderate-High probability of workers receiving a toxic dose if exposed to herbicides during operations for 2, 4-D: aerial, backpack and hack and squirt.	Do not use contract or Forest Service female workers in applications. Require wearing of protective clothing for all Forest Service and contract mixer loaders, backpack applicators, and hack and squirt applicators.
Glyphosate	Broadleaved weeds and grasses	NEG	LOW	MOD INS	MOD	LOW	MOD	MOD	HIGH	MOS =< 100	LOW	LOW	NEG	N/A	LOW	MOD	NEG	N/A	M–I	M	M	A	I	I		
Picloram	Noxious weeds and shrubs	NEG	LOW MOD	MOD INS	LOW MOD	LOW	HIGH	NEG	MOD	— —	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	A	M	M	M	I	I		
Triclopyr	Woody plants and broadleaved weeds	LOW	MOD	MOD	HIGH	MOD	HIGH	MOD	HIGH	MOS =< 100	LOW	MOD	NEG	LOW	LOW	MOD	NEG	LOW	M–I	M	M	A	I	I	Moderate probability of workers receiving a dose that may cause reproductive or developmental effects in operations. Moderate probability of workers receiving a toxic dose if exposed to herbicides during operations for Triclopyr: aerial and hack and squirt.	Do not use contract or Forest Service female workers in applications. Require wearing of protective clothing for all Forest Service and contract mixer loaders, backpack applicators, and hack and squirt applicators.
Dalapon	Annual and perennial grasses and sedges	NEG	LOW	NEG INS	INS	MOD	HIGH	INS	INS	MOS =< 100	MOD	MOD	LOW	N/A	INS	INS	INS	INS	M	M	I	M	I	I	Moderate probability of workers receiving a toxic dose if exposed to herbicides during operations for Dalapon: backpack. Insufficient information on reproductive or development effects for Dalapon.	Require wearing of protective clothing for all Forest Service and contract mixer loaders, backpack applicators, and hack and squirt applicators. Public: Eliminate the use of Dalapon in roadside vegetation management or other uses where members of the public could be exposed to Dalapon either through routine operations or accidents.
Atrazine	Annual grasses	LOW	LOW MOD	MOD	HIGH	HIGH	HIGH	HIGH	HIGH	MOS =< 100	HIGH	HIGH	MOD	N/A	HIGH	HIGH	MOD	N/A	M	M	M	M	M	I	High probability of workers receiving a dose that may cause reproductive or developmental effects in operations. Moderate probability of workers receiving a toxic dose if exposed to herbicides during operations for Atrazine: aerial and backpack.	Do not use contract or Forest Service female workers in applications. No aerial applications. Require wearing of protective clothing for all Forest Service and contract mixer loaders, backpack applicators, and hack and squirt applicators.
2,4-DP	Broadleaved weeds and herbaceous plants	LOW	MOD	MOD	HIGH	LOW	HIGH	LOW	HIGH	— —	NEG	NEG	NEG	NEG	NEG	NEG	NEG	NEG	A	M	M	A	I	I		
Hexazinone	Most broadleaved weeds and grasses	NEG	LOW MOD	LOW INS	LOW MOD	LOW	HIGH	LOW	MOD	— —	LOW	MOD	NEG	N/A	LOW	LOW	NEG	N/A	M	A	M	M	I	I		
Fosamine	Hardwood shrubs	NEG	INS	INS	INS	INS	INS	INS	INS	No information on cancer causing potential	INS	INS	INS	INS	INS	INS	INS	INS	M–I	I	I	I	I	I	Fosamine: There is insufficient information for conducting a full toxicologic evaluation.	Eliminate Fosamine from the Region's program.
Dicamba	Broadleaved weeds and brush	LOW	LOW	LOW INS	HIGH	LOW	MOD	MOD	HIGH	MOS =< 100	LOW	LOW	NEG	LOW	LOW	MOD	NEG	MOD	I	M	M	M	I	I		
Asulam	Bracken fern, broadleaved weeds and perennial grasses	NEG	LOW	MOD	MOD	LOW	MOD	LOW	MOD	— —	LOW	LOW	NEG	N/A	LOW	LOW	NEG	N/A	M	A	M	M	I	I		
Tebuthiuron	Woody range species	LOW	LOW	NEG INS	LOW MOD	MOD	HIGH	MOD	HIGH	MOS =< 100	LOW	MOD	NEG	N/A	LOW	MOD	NEG	N/A	M–I	M	M	M-I	I	I		
Diuron	Grasses and broadleaved weeds	NEG	INS	INS	INS	INS	INS	INS	INS	No information on cancer causing potential	INS	INS	INS	INS	INS	INS	INS	INS	I	I	I	M	I	I	Diuron: Limited data suggest that Diuron has relatively high toxicity and high exposure for backpack application. In addition, there is insufficient information for full toxicologic evaluation.	Eliminate Diuron from the Region's program.
Simazine	Annual grasses	NEG	LOW	INS	MOD	MOD	HIGH	MOD	HIGH	MOS =< 100	MOD	MOD	LOW	N/A	MOD	MOD	LOW	N/A	M	I	M	A	I	I	Moderate probability of workers receiving a dose that may cause reproductive or developmental effects in operations. Moderate probability of workers receiving a toxic dose if exposed to herbicides during operations for Simazine: aerial and backpack.	Do not use contract or Forest Service female workers in applications. Require wearing of protective clothing for all Forest Service and contract mixer loaders, backpack applicators, and hack and squirt applicators.
Bromacil	Broadleaved weeds, grasses and shrubs	NEG	LOW MOD	MOD	LOW	LOW	HIGH	NEG	MOD	— —	N/A	HIGH	LOW	LOW	N/A	MOD	LOW	LOW	M	M	M	M	I	I	Moderate probability of workers receiving a dose that may cause reproductive or developmental effects in operations. Moderate probability of workers receiving a toxic dose if exposed to herbicides during operations for Bromacil: backpack (high) ROW and hack and squirt.	Do not use contract or Forest Service female workers in applications. Require wearing of protective clothing for all Forest Service and contract mixer loaders, backpack applicators, and hack and squirt applicators.
Amitrole	Annual and perennial grasses and herbaceous vegetation	NEG	MOD	HIGH	INS	HIGH	HIGH	INS	INS	High risk for general health, reproductive and fetal health	MOD	HIGH	LOW	MOD	INS	INS	INS	INS	M	M	I	A	I	I	High probability of the public receiving a toxic dose if exposed to Amitrole during routine operations. A probable human carcinogen, high cancer potency.	Eliminate Amitrole from the program. Mitigation measures may not be consistently effective in managing the risks.